

A P H A S I A
AND THE LOCALISATION
OF THE CEREBRAL SPEECH MECHANISM
BY
W I L L I A M E L D E R. M.B., C.M., 1885.



P R E F A C E.

Scope of the Thesis.

It is not my intention in this Thesis to discuss the whole of the disturbances of speech. I intend to limit myself entirely to the central mechanism concerned in speech written and spoken, the disturbances of that mechanism and the localisation of the lesions which produce such disturbances. In other words I shall limit myself entirely to that part of the mechanism situated in the cerebrum. I shall endeavour to show how important a study of aphasia is from a surgical point of view, how much an accurate knowledge by the Surgeon of speech disturbances contributes to the facility of localising lesions in the cerebral hemispheres, and so enables him to judge whether surgical interference could relieve the patient or not, and what form that surgical interference should take.

Aphasia and speech disturbances have I hold been too much considered in the past to be subjects almost entirely for the Physician. Our Text-Books on Surgery have devoted far too little space to the Diagnosis of the different forms of Aphasia and the localisation of

each form. Important as are such diagnosis and localisation to the Physician they are of even more importance to the Surgeon. It is to be remembered therefore that although I shall say little all through the Thesis of the Surgical aspects of the different questions raised, I have borne them always in mind and shall have something to say on such aspects near the conclusion of the Thesis. In treating of the different questions I shall take them in the following order.-

1. A short Historical Review of the contributions to the discussion of the Cerebral disturbances of speech.
2. A short account of the reception, retention, and production of speech.
3. A description of the different routes concerned in the reception, retention, and production of speech, and their localisation.
4. An account of how reception, retention, and production of speech may be interfered with and the localisation of such interference. This will include all the possible varieties of aphasia and their symptoms.

5. The Clinical varieties of aphasia illustrated by cases of my own observation and hitherto unrecorded, and the conclusions drawn from these cases. Special remarks will be devoted to the subject of Functional aphasias, sensory and motor which have not, I believe, received the attention they deserve.

6. A short summary of the disturbances of the music faculty.

7. The importance of Aphasia to the Surgeon.

8. Concluding Remarks.

H I S T O R I C A L.

The power that man possesses of communicating his thoughts to his fellow men by means of language is one of the most characteristic of the many points that distinguish him from the lower animals. That some of the lower animals do possess the power of communicating with each other there can be little doubt, but it can hardly be said of them that they possess the faculty of speech.

Although necessarily experiments in animals can only throw light therefore indirectly on our knowledge of the mechanism of speech in man still such experiments have given a considerable impetus to our knowledge of speech disturbances. That knowledge however has been acquired to a much greater extent by accurate clinical and pathological observation in the human subject. The discussion of speech disturbances has been very intimately associated with the discussion of the question as to whether the different faculties are localised or not localised in the cerebral hemispheres.

Flourens (1) more than fifty years ago stated "that the organ of mind like the mind itself was one and indivisible, there being no differentiation of

function, but each and every part possessed of the potentialities, and capable of exercising every function, pertaining to the whole." These views were accepted for many years and although many facts in cerebral pathology were inexplicable under the non-localisation theory, still observers did not possess sufficient information to upset it altogether. It was not till 1861, when Broca (5) recorded his cases of aphasia localising the lesion to one part of the left hemisphere, that the opinions of the opponents of the non-localising observers began to gain ground.

Previous to Broca's time many theories had been propounded as to the cause of disturbances in speech. Lordat (2) in 1820 who himself became aphasic later ascribed this affection not to paralysis of the tongue but to inco-ordination of the muscles used in speaking. Bouillaud⁽³⁾ following a theory of Gall a few years later located the faculty of language in the frontal lobes of the brain as a result of his clinical observation.

Dax in 1836 (4) from the observation of cases, stated that patients suffering from aphasia along with paralysis were always paralysed on the right side and that therefore the faculty of language was situated in the left side of the brain. G. Dax (son of the

previous) made another step forwards when he located the lesion that causes aphasia in the anterior and outer part of the middle lobe of the left cerebral hemisphere. This was in 1863, two years after Broca had published his two famous cases. The 1st (5) where the lesion was in the left frontal lobe, and the 2nd (5) where it was more limited viz. to the posterior part of the 2nd and 3rd left frontal convolutions.

Clinical observers from this time began to pay particular attention to this region and many cases were published which confirmed more or less the accuracy of Broca's views but as time went on the number of cases which did not conform to the views of Broca also began to accumulate and it became necessary to modify the view that all disturbances of speech were due to lesions of Broca's convolution. Charcot who had produced 10 or 12 successive cases confirmatory of Broca's views gave an account of a case where Broca's convolution was intact but with a lesion in the posterior part of the Island of Reil and the lower marginal convolution. Broca himself had assisted at the post mortem examination of this case and acknowledged the accuracy of Charcot's observations. Cases also began to be recorded where lesions in the right cerebral

hemisphere in the region corresponding to Broca's convolution occurred without disturbances of speech, showing that the speech centres were located on the left side only. To show how difficult it was for clinical observers to accept Broca's views the following quotation is from the famous lectures of Trousseau on Aphasia (6). "You see gentlemen that I have kept back none of M. Broca's arguments and that I have allowed them to be stretched almost to the limits of absurdity, for is it possible in physiology to admit that in an organ so exquisitely symmetrical as the Brain there may be in one of the hemispheres a portion discharging a function which does not appertain to the other hemisphere? Analogy and common sense would protest against such a conclusion, and although in almost all the cases of aphasia which have come under my observation the paralysis (when present) always affected the right side and I was therefore obliged to admit a lesion of the left hemisphere I could not accede to M. Broca's strange doctrine".

Trousseau summarises the position of the subject at that time thus - (16 p. 253). "Aphasia is produced in nearly all cases by an injury to the frontal lobes, as Professor Bouillaud has shown; the

lesion, as Dr Marc Dax has established, is almost exclusively confined to the left hemisphere; whilst its most frequent seat is the posterior part of the 3rd left convolution as M. Broca was the first to point out. Many cases in the next few years were accurately observed and the results of many post mortems recorded in this country, in France and in Germany by such observers as Broadbent (7), Sanders (8), Hughlings Jackson (9), Gairdner (10), Ogle (11), and Bastian (12), and from these it was seen that it was necessary to differentiate several varieties of aphasia.

Wernicke (13) in 1874, published his classical paper pointing out clearly that Aphasia could be divided into two distinct forms, namely motor and sensory. That whilst the first is produced by a lesion in Broca's convolution, the sensory form is produced by a lesion in the first temporal convolution. He also very clearly pointed out several subvarieties of these two principal forms. Thus the upholders of the localisation theory of cerebral functions gradually got stronger evidence from a study of aphasia in support of their position but in the next few years, the question was raised from a position of doubt to one of certainty, because about this time began those

experiments on the brains of the lower animals which are associated with the names of Fritsch and Hitzig, Ferrier, Horsley, Beevor, Semon, Krausse, and Francois Franck.

Cerebral non-localisation may be said to have been finally vanquished at the International Medical Congress in London in 1881 when Goltz of Strasburg (14) the chief of the few remaining champions of the Flour-entian system, produced a dog which he had operated on and "which he exhibited before the physiological world as a practical refutation of the theory of localisation". (1). It was found however at the post mortem of this dog that the cortical areas had not been destroyed so completely as Goltz had believed. At the same Congress, Ferrier produced two monkeys, in one of which he had destroyed the motor areas and there had resulted not only hemiplegia but also late rigidity. In the other the superior temporo-sphenoidal convolution had been destroyed with the result that the animal had been rendered deaf.

Meantime perhaps the ablest monograph up to this time on aphasia was published by Kussmaul (15) in 1877, in which he added another to the chief varieties of aphasia viz.- Word blindness. Both Broadbent (16)

and Wernicke (13) had described cases of this kind but they had not clearly differentiated it from the other varieties.

In 1877 Barlow (55) published his case which proved that although the motor speech centre is situated in Broca's convolution on the left side, it was quite possible when Broca's convolution was destroyed for the corresponding part of the right hemisphere to take up its function. In 1881 Madame Skwortzoff in her able Thesis to the Faculty of Medicine in Paris (59), contributed most valuable observations of cases of word blindness and word deafness. In 1885 Grainger Stewart (31) gave an excellent resume of the subject and in the same year appeared Lichtheim's well known paper (44) in which he differentiated seven different types of Aphasia, and in 1886 Wernicke (47) contributed further observations on the subject. In recent years probably the ablest contributions on aphasia have been published in France chiefly on word-blindness and agraphia by Dèjerine (17), Sérieux (18), and Pitres (19) in which they distinctly prove the existence of two distinct forms of word-blindness produced by lesions in two distinct situations and at the same time rather tending to disprove the existence of

agraphia pure and simple without any other aphasic symptoms. Henschen in 1890 (43) in his excellent work on Cerebral pathology contributed many valuable observations to the Pathological localisation of aphasic lesions. Wyllie in 1894 (20) in his able work on Disorders of Speech produced the most accurate and comprehensive account of the whole subject that has yet been published. In that work the contributions of the French School to the subject of word-blindness were brought clearly before the profession in this country, and an article by Hinshelwood (53) on word-blindness and visual memory contributed recently (Decr. 1895) to the Lancet besides again bringing the work of Dèjerine, Sérieux and Henschen on word-blindness before the English profession added several, more important observations to those already published.

II. THE RECEPTION, RETENTION & PRODUCTION OF SPEECH

In considering the mechanism of speech it is

perhaps best to study it not as found in its more perfect form in the adult but rather to study it in the child who is acquiring it. The child hears words and understands the meaning of those words for a considerable time before it is able to produce words. The process therefore of reception goes before that of production of language. And here I wish at once to make it clear that language in this sense means all the methods by which man is enabled to communicate his thoughts to his fellowman. Speech written and spoken is of course the most commonly used of all these methods, and is therefore taken as the type, but the other methods such as by signs and by muscular movements as used by the deaf, and by tactile sensations as used by the blind, have an analogous mechanism called into action. The acquiring and production of music is an analogous process to that of the acquiring and production of language and it also will receive some consideration in this paper.

The means by which speech is acquired is by the sensory organs. The child first gets its knowledge of language by the ears. The mother speaks and the child hears the sound and gradually gets to understand the meaning of that particular sound after it has been

repeated on several occasions. It is enabled to do so by means of memory. How is this brought about? It is brought about by the next process in learning a language viz. what I have called retention of speech or storing up of the sound images of the word spoken. Now it is generally supposed that, on receipt of sensory impressions whether from ordinary sensory nerves, from the muscular sense or from the special senses, an effect is brought about in a particular cell or cells of the cerebrum, which is lasting, and that effect can either be revived from within the brain or from without on any future occasion. This recalling or reviving is therefore the memory for the past sensation. One of the best ways to recall this past impression is to repeat a similar impression when at once the memory of the previous one is recalled. The repetition of the same word to the child therefore recalls to its memory the previous word and at the same time the associations of the previous word and it is so enabled to learn the meaning of the word. After it knows once the meaning, such meaning and its associations become imprinted in the cells of its cerebrum, so that in the next repetition of the word the child recalls the meaning much more easily. It

is easily therefore, understood how repetition of words and repetition of the circumstances which recall their meaning or repetition of the meanings themselves make the individual gradually more expert in the recognition of words and in the recognition of their meaning. And what applies to hearing and hearing words applies to all the other sensations and sensory impressions which it is possible for the brain to receive, as for instance, when we see anything once we probably remember it and recognize it on seeing it a second time, but after seeing it on many occasions we recognize it much more readily. Later on I shall probably have to refer to memory for muscular movements. The same process holds good for them and probably is brought about by sensory impressions also as Bastian holds.

Again going back to the case of the child. The mother speaks to it and it gradually stores up in its cerebral cells the word sounds which it hears and gradually recognizes the sound of one word after another, and the meaning associated with such sounds. The next process is the production of language. I shall not enter here into an account of the interesting process that most children go through in the

production of a language for themselves. They however gradually give up that baby language and by imitating those who speak, they are enabled to reproduce the same sounds which they have heard others produce and the associations of such sounds are recalled just as if another had produced them. Now a child is enabled to learn to speak from seeing the movements of the mouth, lips etc. of those who speak to it. It probably does not notice each individual movement and try to imitate each movement but it sees that sounds are produced by movements. It attempts to produce a particular sound and it is enabled to correct itself by means of its hearing and by gradual imitation, it pronounces the words correctly. Deaf mutes are dumb because they are primarily deaf. They have never heard sounds, and therefore, they never try to produce them, but that they can produce them is shown by the fact that they can by perseverance be taught to speak. From what I have already said it will be seen that sight plays also a part, although perhaps not so important a one in the acquiring of spoken speech. But sight plays a much more important part in the acquiring of the other forms of speech later on in the life of the child. Written and printed speech

is acquired primarily by means of sight. The child sees a particular form or sign - viz. a letter. He associates it with a particular sound by hearing it pronounced. He gradually puts one or two letters together and joins the one sound to the other to form a word with its associated meaning, and these sight impressions are stored in the cerebral cells, are linked to the sound impressions also stored in the cerebral cells, and can be recalled by memory either from within or from without, from hearing or from seeing. As the impressions of sounds are received, stored up and produced by a child, so the impressions of these signs or letters written or printed are received, stored up, and produced by the child.

The production is done by imitation, analogously to learning to speak, but here it is a case of copying the form of the letters, in other words learning to write. The child imitates the design of the letters, tracing them bit by bit, but through time by means of memory of past attempts he becomes more expert until he is able to write proficiently.

A blind agraphic patient is thus analogous to a deaf mute. Is there any case where a child was a blind mute? I have not seen any notice taken of such

a condition, but I think that if a case which Dr Broadbent (7) published as a case of Congenital Aphasia be studied, it will be seen that the boy was a blind-mute. He was not able to speak although he could hear but he could not see letters. The case was published before word-blindness had been described, and perhaps it is too much to draw my conclusion from description only. It will thus be seen 1st. that speech is received by the brain by two routes - viz. by the ears and auditory nerves and by the eyes and optic nerves. 2nd. That these sensory impressions are stored up in the memory and are associated so that the memory of one sensory impression of the one can recall the memory or sensory impression of the other. For instance, when the letter O is seen, the sound of O is at once recalled in the visual memory. Or again when one thinks of the sound of O, that is, recalls it to memory, the sign O at once is revived in the visual memory and vice versa. 3rd. That speech is produced by the brain by two routes - viz. by speaking and by writing.

It is also to be understood of course that language that reaches the brain cells by sight can be produced by either production route and language

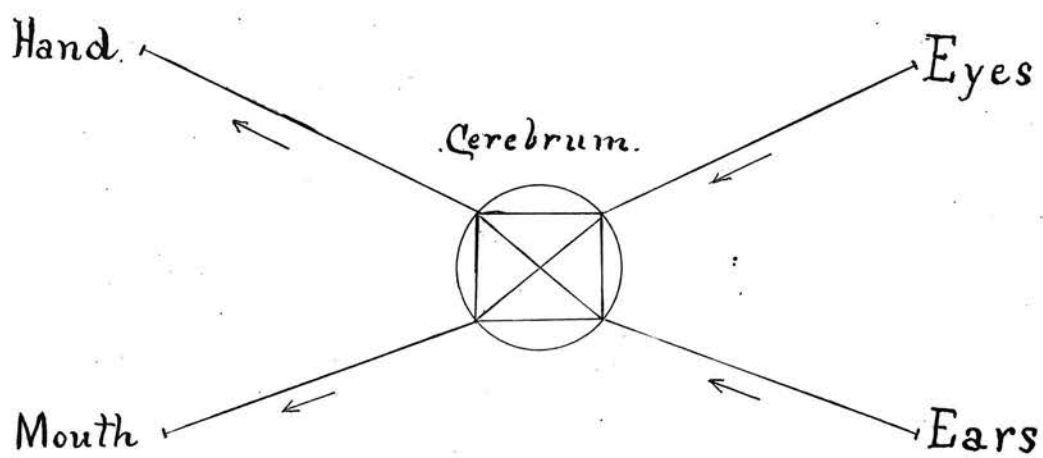


Fig. I.

received by hearing can be produced by either production route. This can easily be indicated by means of a simple diagram. See Fig. I.

These are the usual routes used, but other not so common routes can be and are cultivated as for instance by means of other signs than those of printed or written symbols, as by gestures where the reception route is the same viz.- that of sight, but the production may be done by the whole body or any part of it. Deaf mutes are taught to speak, reading the lips, and hearing is not required for this process. A much more marked example, however, of change of route is the teaching of the blind to read by means of the tactile and muscular sensations of the fingers. The sensory nerves of the fingers (tactile and those of the muscular sense) are here used instead of the optic nerves, but the production route is the usual one, - viz the route used in ordinary oral articulation.

Such is a short outline of the method by which speech is acquired, the memory of speech retained, and revived in the memory at a later period so that it can be reproduced either by speaking or by writing. It is generally believed that the same cells which receive the impressions must be called into action in the

reviving of such impressions, and that the process of reviving in each cell probably consists in a repetition of the original process in that cell. This view was well put by Bastian (61) in his able contribution on the muscular sense where he stated that "when past impressions are revived as ideas or recollections, precisely the same parts of the hemispheres, the same nerve fibres, and the same nerve cells must be called into activity as were previously concerned in the perception of the original impression."

III. RECEPTION, RETENTION and PRODUCTION SPEECH ROUTES.

Having, therefore, given an outline of these processes, I shall now proceed to give an outline of the different routes in the brain concerned in them. I shall look at this part of the subject in the meantime entirely from an anatomical, physiological, and pathological point of view.

I. AUDITORY ROUTE.

And first then let us trace the auditory route from the ear to the auditory centres in the cerebral hemispheres. The tract of this, as well as of the other cranial nerves has been studied very carefully in recent years, experimentally and microscopically, as well as clinically, by such well known observers as Flechsig, Meynert, Bruce and Hans Held, and its course is now pretty well made out. It has been found that the auditory nerve really consists of two parts, one part which originates in the vestibule in the semi-circular canals is the nerve of equilibration. It becomes the anterior root of the auditory nerve and goes to the cerebellum and need not further be considered here.

The posterior root or auditory nerve proper originates in the organ of Corti in the cochlea, passes into the bulb to the lateral tubercle and accessory nucleus of the same side, then passes to the opposite side and by way of the fillet, posterior quadrigeminal body, internal geniculate body and cerebral radiations to the posterior $\frac{1}{2}$ or $\frac{2}{3}$ of the 1st and 2nd temporo-sphenoidal convolutions.

It has been shown experimentally by Ferrier (21) that the position of the centre for hearing is in the 1st temporo-sphenoidal convolution as is indicated in the accompanying diagram. See Fig. II.

Ferrier by stimulating that particular region proved that the animal behaved just as if it had heard a sound in the opposite ear, and by extirpation of the same region in monkeys and other animals he rendered the animals deaf in the opposite ear. He also showed that extirpation of that region on both sides produced total deafness. Schäfer and Sanger Brown from their experiments on six monkeys disputed Ferrier's results, but Ferrier (22) on repeating on a monkey his experiment of bilateral extirpation of the superior temporo-sphenoidal convolution showed that the animal was at first completely deaf and later that although it was not insensible to sonorous vibrations it was quite indifferent to sounds that formerly used to recall some meaning to it. Munk placed the position of the Auditory centre slightly lower than Ferrier,

Clinical evidence on this subject has also shown that the centre is in this region. Mills (23) records the case of a man, deaf for 30 years, whose

brain showed atrophy of the superior temporo-sphenoidal convolutions on both sides and particularly the left. Wernicke and Friedlander (24) have published a case of a woman who as a result of an apoplexy had right hemiplegia, word deafness, aphasia, and paraphasia and a few months later had a second attack affecting the other side with the result that she was rendered completely deaf. At the post mortem both superior temporo-sphenoidal convolutions were affected. Mills (25) records another case, in which a patient, 15 years before her death had an apoplectic attack which left her word deaf but not deaf to sounds or music. Six years afterwards she had another attack, which, however was on the other side as she had left sided hemiplegia chiefly affecting the arm, and became now almost totally deaf. The post mortem revealed the presence of a destructive lesion of the 1st and 2nd temporo-sphenoidal convolutions on both sides, thus accounting for the symptoms during life and confirming the localisation of the auditory centre to this region a region slightly greater in extent than that marked out by Ferrier (2) from his experiments on monkeys.

From what has been already said it will be seen that there is a cortical auditory centre in both

hemispheres and experiments have shown that the ears are bilaterally represented. How this is brought about ~~it~~ is not exactly known but probably there is an arrangement of the auditory fibres much like what will be seen later on in connection with the optic fibres viz. a semi-decussation. Ferrier who is an upholder of this view says that "unilateral extirpation never gives rise to permanent deafness of the one ear but though I have, on many occasions, after extirpation of the auditory area in one hemisphere, observed loss or impairment of hearing in the opposite ear, I have never been able to detect the slightest impairment of hearing in the ear of the same side." Clinical evidence from a study of word deafness and cortical lesions such as give rise to hemiplegia would lead us to the same conclusion because it is necessary for a lesion to destroy the centres in both hemispheres in order to render either ear totally deaf, although as we shall see later the centre for word hearing is situated in the left hemisphere only. This is just what would have been expected from a study of the principle involved in the hypothesis of Broadbent ((26) and (7)) which has been called Broadbent's law and is generally recognised to hold good with regard to the

motor centres and their innervation but which is equally applicable to the sensory centres. It was propounded in 1866 by Broadbent before the experiments on animals had shifted the motor centres from the basal ganglia to the cortex and Broadbent's words have to be considered in that light, but they are sufficiently clear. "Where the muscles of the corresponding parts on opposite sides of the body constantly act in concert and act independently, either not at all or with difficulty the nerve nuclei of these muscles are so connected with commissural fibres as to be pro tanto a single nucleus. This combined nucleus will have a set of fibres from each corpus striatum, and will usually be called into action by both, but it will be capable of being excited by either singly, more or less completely according as the commissural connection between the two halves is more or less perfect." Again in 1872 (7 p.189) he states his hypothesis very distinctly with regard to the special senses. "But there is a peculiarity in the nerve-nuclei or sense centres of the special senses, they are bilaterally associated. This at least is especially the case with the two intellectual senses of sight and hearing, and offers an explanation of the absence of unilateral

blindness or deafness in hemiplegia with marked loss of common sensation in the limbs and trunk. The fused nerve nuclei will constitute a common sense centre which will send up fibres to each half of the cerebrum and thus impressions will travel equally to the two hemispheres etc." In considering the motor side of speech disturbances I will probably have to refer to this law again. The conclusions we can draw therefore from the facts stated in connection with the localisation of the auditory centre in man are.-

1. That the auditory centre is situated in the 1st and 2nd temporo-sphenoidal convolutions. See Fig. 2.
2. That there is a centre in both hemispheres.
3. That each ear is bilaterally represented.

We shall at this point leave the Auditory nerve in the meantime and shall next take up the second route by which sensory impressions of language reach the brain viz. the visual route.

II. VISUAL ROUTE.

If the course of the auditory route has given rise to much discussion, that of the visual has given

rise to even more. It will be convenient to trace the route from the eyes to the visual centre in the meantime just as has been done in the case of the auditory nerve. The optic nerve begins in the retina and passes to the optic commissure or chiasma, where there is a semi-decussation of the fibres. The decussated fibres along with the fibres that have not decussated are then continued in one bundle as the optic tract. This tract winds round the cerebral peduncles and divides at the postero inferior part of the optic thalamus into two bundles of fibres. A small internal bundle goes to the internal geniculate body and the posterior quadrigeminal body. The larger more external passes into the external geniculate body, the anterior quadrigeminal body, and the posterior part of the optic thalamus. From these three masses of grey matter springs the large bundle of fibres which goes by the name of the optic radiations or the radiations of Gratiolet. These fibres pass into the posterior part of the internal capsule pass backwards external to the posterior horn of the lateral ventricle and end in the cortical region of the occipital lobe, chiefly in the cuneus and the most posterior part of the lobe (Dèjerine 17). The

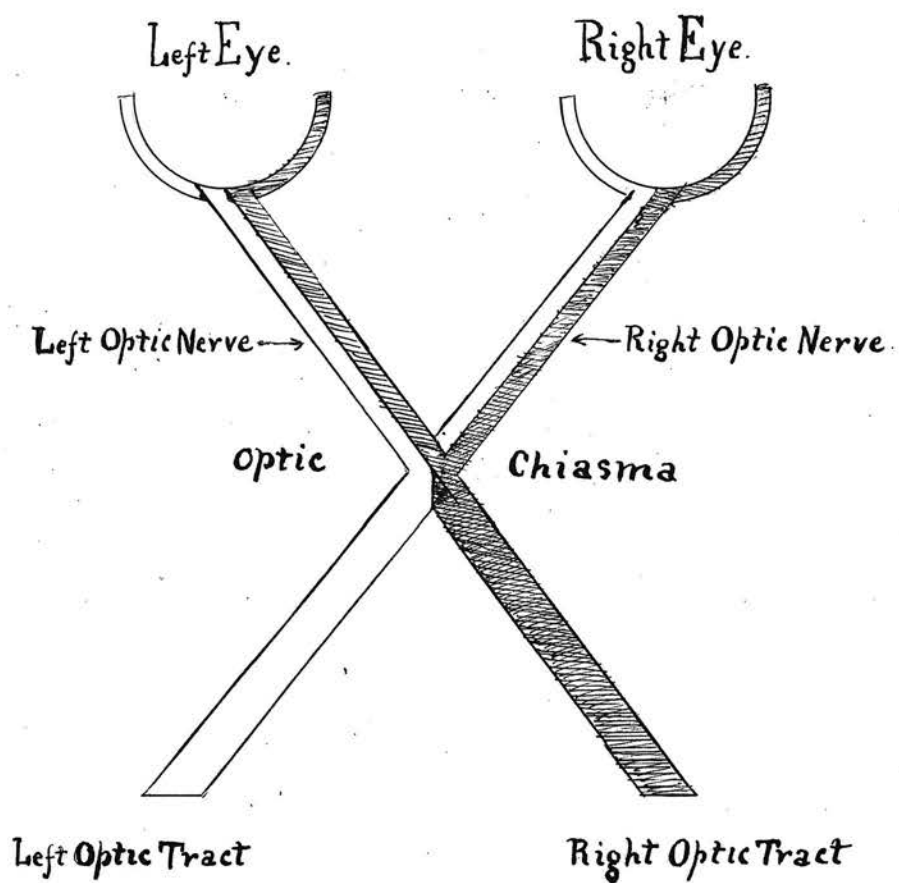


Fig. III.

arrangement of the fibres in the optic commissure has been pretty well made out. It was held by Brown Sequard and others that there was complete decussation at the chiasma but it has been distinctly proved in recent years that there is only a semi-decussation. It has been found that the optic fibres of one half of the retina of each eye cross to the opposite side whereas the optic fibres from the other half of the retina do not cross over. The fibres from the internal or nasal half of the retina cross over whilst the fibres from the temporal or external half of the retina do not cross over but remain on the same side. This can be shown by a simple diagram. See Fig. III.

The result of this is that the optic fibres which physiologically act together from the half of each retina pass into one optic tract, whilst the fibres from the other half of each retina pass into the other optic tract. That is to say, the fibres from the temporal side of the left eye and those from the nasal side of the right eye pass into the left optic tract, whilst the fibres from the temporal side of the right eye, and the nasal side of the left pass into the right optic tract. In the commissure in addition to these fibres there is another bundle of fibres viz. the inferior commissure or commissure of Gudden, a bundle which is purely

commissural. It is situated in the posterior angle of the chiasma, passing along one optic tract it crosses along the posterior part of the chiasma to the optic tract of the other side, and forms the small internal bundle previously mentioned, which goes to the internal geniculate body and the posterior quadrigeminal body. These fibres are simply commissural for these two bodies on each side and have been traced from them to the temporal lobes, (Monakow 27) and need not further be considered here.

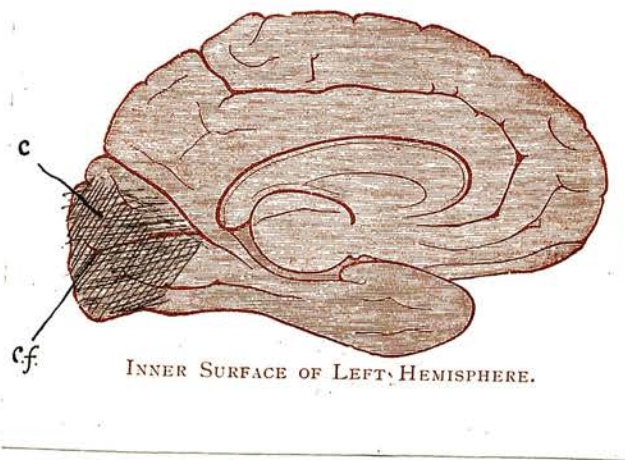
It has been shown experimentally that whilst division of one optic nerve produces complete blindness in one eye, division of one optic tract produces blindness in one half of each retina viz. what is called homonymous lateral hemianopia. If the left optic tract be divided, left lateral homonymous hemianopia is produced, that is to say the temporal side of the left retina and the nasal side of the right retina are rendered blind. As rays of light from the right side of the patient fall on that half of the retina in each eye the patient is thus blind to objects in his right field of vision. He is said to have right lateral homonymous hemianopsia. Hemianopsia being the term applied to the field of vision

whilst hemianopia is applied to the retina itself.

The same symptoms are produced by division of the fibres in any part of their course from the right optic commissure to their termination in the cortex of the occipital lobe. It has been found also that extirpation of the eyeball is followed by ascending degeneration in the optic nerve of the same side and of half of the optic tract as far as the external geniculate body, the anterior quadrigeminal body and the optic thalamus but it does not extend to the optic radiations. Excision of both eyes is followed by ascending degeneration of both tracts as far as the same ganglia (Monakow 27), so that these ganglia are the first visual centres. On the other hand a descending degeneration of these fibres follows lesions of the cortical terminations of the radiations of Gratiolet. The fibres in the optical radiations and also the posterior part of the optic thalamus degenerate and in young animals, and sometimes in man such degeneration extends to the external geniculate and quadrageminal bodies, the optic tracts and optic nerve of the same side as well as the optic nerve of the opposite side. (Monakow quoted by Dèjerine 17). Much discussion has taken place in recent years as to

the precise position of the centre for sight. Ferrier as result of his experiments located it in animals in the ^{ul}angular gyrus. Munk, on the other hand, stated that the visual centre was in the outer convex part of the occipital lobe. Horsley and Schäfer (quoted by Mills 28) found that extensive lesions both of the occipital and the temporal lobes were invariably followed by visual disturbances taking the form when the operation was confined to one side of the brain of bilateral homonymous hemianopsia but in nearly every case the hemianopsia was merely temporary. The most marked results were obtained when the occipital lobes were the seat of operation, extensive unilateral lesions producing amblyopia but only temporary. Schafer and Sanger Brown in their experiments destroyed both angular gyri without any change in vision. It has been found that both Ferrier and Munk were right in their localisation because the primary perceptive visual centre has been proved to be in the occipital lobes, and the specialized visual centre viz. that for words, signs etc., in the angular gyrus.

In recent years from a careful study of clinical pathological and experimental evidence the primary visual perceptive centres have been localised in man



cf = calcarine fissure.
c = cuneus

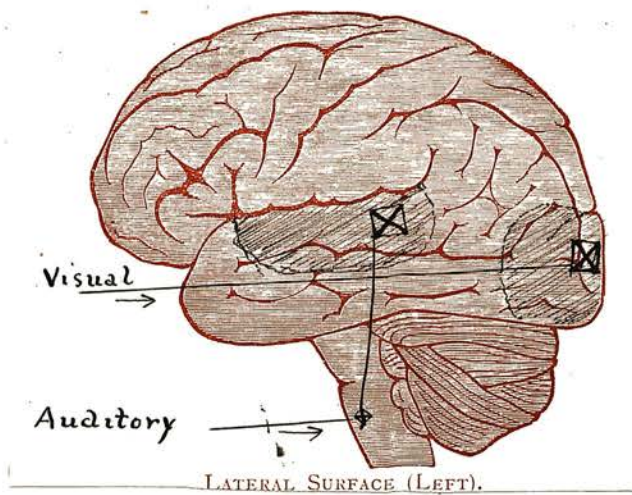
The shaded portion = The visual perceptive centres

Fig. IV.

chiefly to the internal surface of the occipital lobes. Henschen (29) from a careful study of a large number of cases arrives at the conclusion that only part of the radiations of Gratiolet are visual and part have to do with the reflexes of the eye. The visual part is ventrally situated in the radiations and goes to the neighbourhood of the calcarine fissure. He holds that a lesion in this neighbourhood produces complete hemianopsia. and quotes one of his cases where the cortex and only the cortex in this region was involved with complete hemianopsia. He also believes that the retinal field can be divided into different quadrants, each having its special representation in the optical radiations and in this cortical region, and contrary to the opinion of Ferrier who locates it in the angular gyrus he holds that the macula is represented here also. Vialet (30) also agrees with Henschen that the calcarine fissure is about the middle of the cortical visual centre of man. The position of the centre is seen in the accompanying Diagram. Fig. IV.

The conclusions to be drawn therefore from a consideration of all the evidence on visual centres are these.-

- 1st. That the perceptive or primary visual centre in man is in the cortex of the occipital lobe and most probably in the internal surface of



Visual and Auditory Centres

Fig. V.

that lobe in the immediate neighbourhood of the calcarine fissure.

2nd That there is a centre in both hemispheres.

3rd That one half of both retinae is represented in each hemisphere.

4th That as both eyes act together there are probably commissural fibres connecting the centre in one hemisphere with the centre in the other hemisphere through the corpus callosum, but these have not yet definitely been made out.

The accompanying diagram (Fig. V.) shows the relative position of the two primary perceptive centres and it will now be convenient to take up the consideration of the two routes by which speech is produced by the brain before discussing the means by which these centres are linked together and in what way speech is able to be received and transmitted from the sensory or receptive centre to the motor or productive centres.

III. MOTOR SPEECH ROUTE.

And first then let us take up the route concerned in the production of spoken speech. In the act of speaking there are three distinct mechanisms called into action viz. The respiratory, the vocal,

and the oral articulative. A blast of air is forced by the expiratory muscles through the trachea and larynx and the vocal mechanism acts so as to produce voice or sound by means of movements of the vocal cords. The voice laden air passes into the pharynx and mouth and is there acted on by the oral articulative mechanism, so as to produce words.

It is therefore necessary in order to a proper understanding of the speech production route to trace the innervation of these three mechanisms. I have elsewhere (60) entered into a discussion as to the cerebral localisation of the respiratory centre, and shall only say here that although it is believed that the respiratory mechanism has a cortical centre the precise position of it has not yet been made out. Following Broadbent's law, however, it must be bilaterally represented and therefore must be very rarely completely disorganised. As respiration requires to go on continually in order that the individual may live there is a primary centre in the bulb which carries on the usual respiratory movements necessary for life. In the paper quoted I have given some reasons for supposing that the cortical centre for the respiratory movements for speech is situated in the

motor areas, but evidence on this point is still very much wanted.

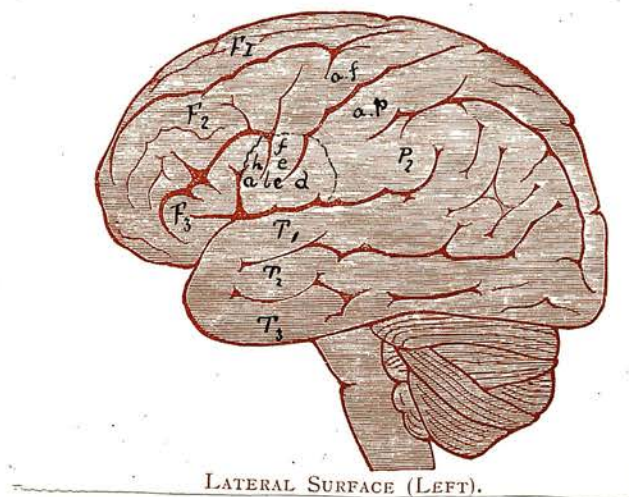
On the other hand experimental physiology has done much in recent years to locate exactly the position of the centres for the vocal mechanism as well as the oral articulative mechanism. Before the days when Fritsch and Hitzig began their experiments on the brains of living animals, the motor centres were generally believed to be in the basal ganglia, but since that time the researches of Ferrier (21) Horsley and Beevor (32), Horsley and Semon (33), Krausse (34), Francois Franck (39) and Risien Russell (33) have completely established the fact that the motor centres are situated in the neighbourhood of the fissure of Rolando.

Amongst the muscles whose centres have been so localised are those concerned in the production of speech both spoken and written.

Ferrier (21) locates the centres "for the lips and tongue as in articulation" in the foot of the third frontal convolution, the foot of the ascending frontal and the foot of the ascending parietal, whilst Horsley and Beevor (32) and Horsley and Semon (33)

locate the centre for these movements in the same region, but they do not include the posterior part or foot of the third frontal in this centre. Horsley and Semon (33) showed very clearly that the anterior part of the foot of the ascending frontal convolution Fig. VI (b) was the centre for adduction of the vocal cords and that they were completely bilaterally represented in the hemispheres so that stimulation of this region on one side produced adduction of both, and that destruction of this region on one side did not produce paralysis of the adduction movements of any side. Several cases have been published against this view in which lesion of this area on one side is said to have produced paralysis of one vocal cord. Such cases have been published by Dèjerine (36) by Garel (37) and by Garel and Dor (38).

Semon (40) however pointed out that these observations cannot be reliable as lesion of the whole of this region as in hemiplegia never produces paralysis of one vocal cord. Recently Risien Russell by very careful experiments has located the centre for abduction of the vocal cords, not far from this same area viz. about (h) Fig. VI, but he found that the



LATERAL SURFACE (LEFT).

a = psycho-motor speech area.
b = adductor centre
cd = oral articulative centre
e = lower face centre
f = upper face centre
h = abductor centre

Fig. VI.

adductor centre was much more active than the abductor, and hence the difficulty in differentiating the two centres. Horsley and Beevor (32) found that the remainder of the lower part of the ascending frontal convolution as well as the lower part of the ascending parietal was the centre for movements of the tongue and throat and for opening and closing the mouth (c.d.) and that immediately above these areas, was an area for the lower face and angle of the mouth, and higher up still a centre (f) for the upper part of the face (see Fig. VI.) These were the positions of the centres in the anthropoid apes and higher animals and it was generally supposed that the foot of the third frontal and the cortex immediately behind were the parts of the human brain which corresponded to these centres, and the oral articulative mechanism was therefore supposed to have its centre in Broca's convolution, and hence lesion of this region on the left side was supposed to produce always motor aphasia. Wyllie (20 page 301) pointed out that whilst Ferrier probably from clinical as well as experimental evidence included the foot of the third frontal in the oral articulative centre, other observers did not, and

and he therefore threw out the hypothesis that the foot of the third frontal is the centre for the psychomotor images of speech, lesion of which produces motor aphasia whilst the oral articulative mechanism has its centre in the lower part of the ascending frontal and the lower part of the ascending parietal (b.c.d.e).

In the Edinburgh Hospital Reports for 1895,⁽⁶⁰⁾ I published a case which went a long way to confirm the hypothesis of Wyllie, and as the subject is one of considerable importance I give a note of the case here.

CASE I. (Personal Observation). Thomas T.

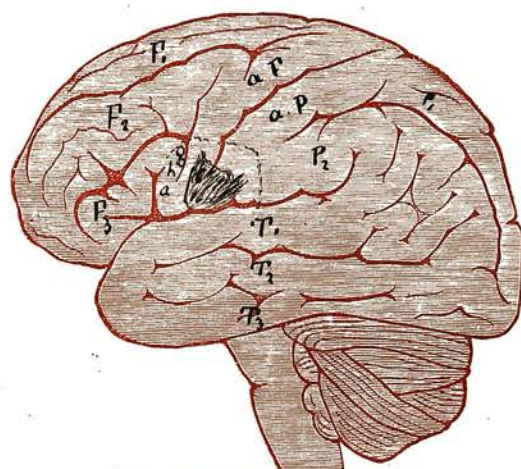
aet 60 admitted into Leith Hospital 16th May 1895 complaining of difficulty in speaking. The day before his admission his speech suddenly became indistinct and blurred and it was observed that the saliva trickled from his mouth. This appears to have been the only symptom. He went to work but returned home at mid-day not feeling able for it. Next day he was no better and he asked assistance in putting on his coat. He walked to the Hospital, about 300 yards, and was admitted. On admission patient looked feeble, rather stupid and dazed, but conscious and understood all that was being said. The saliva was trickling from

the mouth. There was paresis of the right side of the face and angle of the mouth, not involving the orbicularis palpebrarum, the patient being able to close his eyes quite well. He spoke in a very thick blurred manner so that it was difficult for him to pronounce the words distinctly. This, however, was seen to be due simply to a difficulty he had in moving the tongue, lips and other muscles of articulation as readily as he wished to. There was no real aphasia. He knew what was being said, he knew what he was going to say, tried to say it and always succeeded in saying it but the words were blurred and indistinct. He had difficulty in swallowing even liquids. His voice was unimpaired; there was no hoarseness or other indication that the movements of the vocal cords were in any way affected. He could protrude his tongue although it came out rather slowly. It did not point distinctly to either side. There was no paralysis of the arm or leg. Sensation remained unimpaired. On the 19th the general appearance of patient was a little better speech thicker, trouble in swallowing considerable, facial paresis present. The patient gradually got weaker without showing any new symptoms except dilatation of the right pupil and general livid flushing on

the right side of the face, and hypostatic congestion of the lungs. On the 22nd he became comatose and died.

The diagnosis was made of a lesion in the white substance of the left hemisphere cutting off the fibres passing from the cortex in the lower part of the fissure of Rolando to the internal capsule.

The result of the post mortem showed that not only were these fibres involved but the cortical substance itself at the lower part of the ascending frontal and ascending parietal convolutions was destroyed without producing aphasia. The following is the post mortem report;- on removing the skull-cap the dura was not adherent, the veins on surface of the brain were seen to be full. In the lower part of the ascending frontal convolution about half an inch from the sylvian fissure a blood clot was seen which had just pushed its way through the cortex at that particular spot, and it could be seen before cutting into the hemisphere, that the cortical substance all around that spot was quite thin most of it having been destroyed. On slicing the brain horizontally, a blood clot of about a dessert spoonful in quantity was found at the level of the lower part of the ascending frontal and ascending



LATERAL SURFACE (LEFT).

Case. I.

The shaded part shows the position
of the haemorrhage

Fig. VII

parietal convolutions. It had destroyed almost entirely the cortical substance of the lower end of these convolutions from the sylvian fissure to as high up as the level of the fissure that divides the second from the third frontal convolution. It extended a little higher up in front than it did behind. It did not quite involve the whole of the lower end of the ascending frontal as it did not quite reach the precentral sulcus, there remaining intact a strip of cortex adjoining the foot of the third frontal The foot of the third frontal also remained intact. The island of Reil also was not involved. The area of the cortex involved is seen in the accompanying diagram, Fig. VII.

Internally the haemorrhage extended inwards and forwards immediately above the level of the lenticular nucleus in a very thin layer for about two thirds of the distance between the cortical surface and the internal surface of the hemisphere. This layer was for about half of its extent no thicker than a penny. Its furthest internal point was in close relation to the anterior limb of the internal capsule, and quite in front of the motor tract.



Case. I.

Photograph of Left Hemisphere

The upper part of the Hemisphere has been reflected towards the middle line and supported.

Position of Haemorrhage seen in Horizontal section

Fig VIII.

Fig. VIII. is a photo of the brain in section showing the position of the haemorrhage.

There was no lesion in the pons nor any other part of the central nervous system. Since publishing the above I have come across a case much similar. In the recently published Text Book on Nervous Diseases by American authors, edited by Dercun 1895, Mills quotes a case (page 409) observed by himself as confirmatory of Wyllie's hypothesis - he says;- "In a case of typical orolingual paralysis recorded by me, the patient had distinct facial paralysis in the muscles supplied by the lower distribution of the seventh nerve, and also lingual paresis; probably also slight want of control over the right orbicularis palpebrarum. He had some power over the nasal dilator and good control of masseter, pterygoid and temporal movements. Articulation was distinctly involved because of orolingual mono-paresis. He could talk but pronounced certain words indistinctly. He had no difficulty in propositionizing (that is, there was no true motor aphasia W.E.). A focus of strictly cortical yellowish softening was found involving the lower extremities of both central convolutions (the ascending

frontal and ascending parietal W.E.) both on their external and Sylvian surfaces, and a soft one half an inch in diameter about the middle of the internal portion of the insula. The softening reached into the central fissure thus taking in a posterior, inferior strip of the second frontal convolution. Its greatest height was one and a half inches upward from the Sylvian fissure, its width along this fissure one and a quarter inches. The anterior limit of the lesion was a fourth of an inch caudad of the presylvian fissure. This case is so very like my case in the position of the lesion and the symptoms that I find that Mills draws almost the same conclusion from his case that I did from mine, viz.- that the psycho-motor speech area, or as he calls it following Broadbent the propositionizing centre, is in the foot of the 3rd frontal, whilst the vocal and oral articulative centres or as he calls them the utterance centre are situated in the lower part of the ascending frontal and lower part of the ascending parietal convolutions. Utterance centre although a convenient term is not, I think, a physiologically accurate one because I believe there are several centres in this area and not only one utterance centre. There is the centre for adduction

and the centre for abduction of the vocal cords, the centres for movements of the tongue, throat, lips, and cheeks, and these centres are quite capable of acting independently, but in speech production they are acted on by the psycho-motor or propositionizing centre in the foot of the 3rd frontal, which centre does all the cell grouping or regulating, or co-ordinating of these centres so that the correct word or words are produced.

Wyllie's terms therefore of psycho-motor for the propositionizing centre and executory-motor or vocal and oral articulative mechanism for the utterance centre, are more physiologically correct.

Granted that these two centres exist then they must be joined together by connecting fibres which in all probability lie either immediately beneath the cortex or in the cortex itself. As it is known that corresponding parts of the cortex of each hemisphere are also linked together the foot of the 3rd frontal on the left side is joined to the corresponding part on the right side by commissural fibres through the corpus callosum. The lower parts of the ascending frontal and ascending parietal convolutions for the

same reason are connected with the corresponding parts on the opposite side by commissural fibres also through the corpus callosum. It may be by means of these fibres that there is a more or less bilateral representation, but it is possible that the bilateral representation may be partly, if not wholly, brought about by commissural fibres in the bulb. The importance of these commissural fibres will appear when lesions in this region are considered. Tracing now the course of the motor speech route from the propositionizing or psycho-motor centre in the foot of the left third frontal, the nervous energy passes posteriorly to the vocal and oral articulative centres on the same side where the proper cell grouping takes place for the production of the correct words. The vocal and oral articulative mechanism centres on the opposite side must also be acted on by the same psycho-motor centre in the same way and at the same time, but in what way the nerve influence reaches the right side has not hitherto been known. Besides proving the precise position of the vocal and oral articulative centre the case I have published, as also Mill's, proves that the energy to reach the right side either

passes from the foot of the 3rd left frontal to the foot of the 3rd right frontal and thence to the vocal and oral articulative mechanism on the right side or what is not so likely it passes from the 3rd left frontal directly to the vocal and oral articulative centres on the right side, or what is least probable of all it passes from the psycho-motor centre on the left side to the vocal centre on the same side and then to the vocal and oral articulative centres on the opposite side. I draw this conclusion because the lesion in my case of destruction of the left oral articulative centre (the vocal centre escaped destruction) produced only mono-paresis of the oral articulative mechanism and not complete paralysis as would have been the case if the whole of the fibres from the foot of the 3rd frontal on the left side had to pass through this destroyed centre first before reaching the other side. The case would have shown all the symptoms of a sub-cortical motor aphasia instead of simply being one of dysarthria, if this conclusion is not correct. That these commissural fibres high up in the cerebrum are of great importance is also seen from the fact that lesion in the fibres from the oral articulative centre on the right side produces the

same symptoms as the same lesion on the left side viz. dysarthria, showing that the commissural fibres in the pons are not quite sufficient for the innervation of both sides. If these latter commissural fibres were sufficient, then lesion of the fibres on any side above the pons would not produce any paresis of the oral articulative mechanism because the nervous energy could reach the pons by the side which remained intact. It has been found, however, that more nervous energy passes down the fibres on the left side than on the right as lesion on the left side produces more marked dysarthria than the same lesion on the right side, and also that a right sided lesion is sooner recovered from than a left sided one. Lichtheim (44 page 480) who of course treats Broca's convolution as one centre and not as containing several centres shows that there must be decussating fibres crossing high up in the cerebrum. He says.- "Now if the fibres from Broca's centre reached the basal organs down the left side only the usual persistent aphasic symptoms would arise from lesion of the left peduncle or internal capsule as uniformly as they do from those of the centre itself. But we know that this is only exceptionally the case, hence there must be a partial decussation.

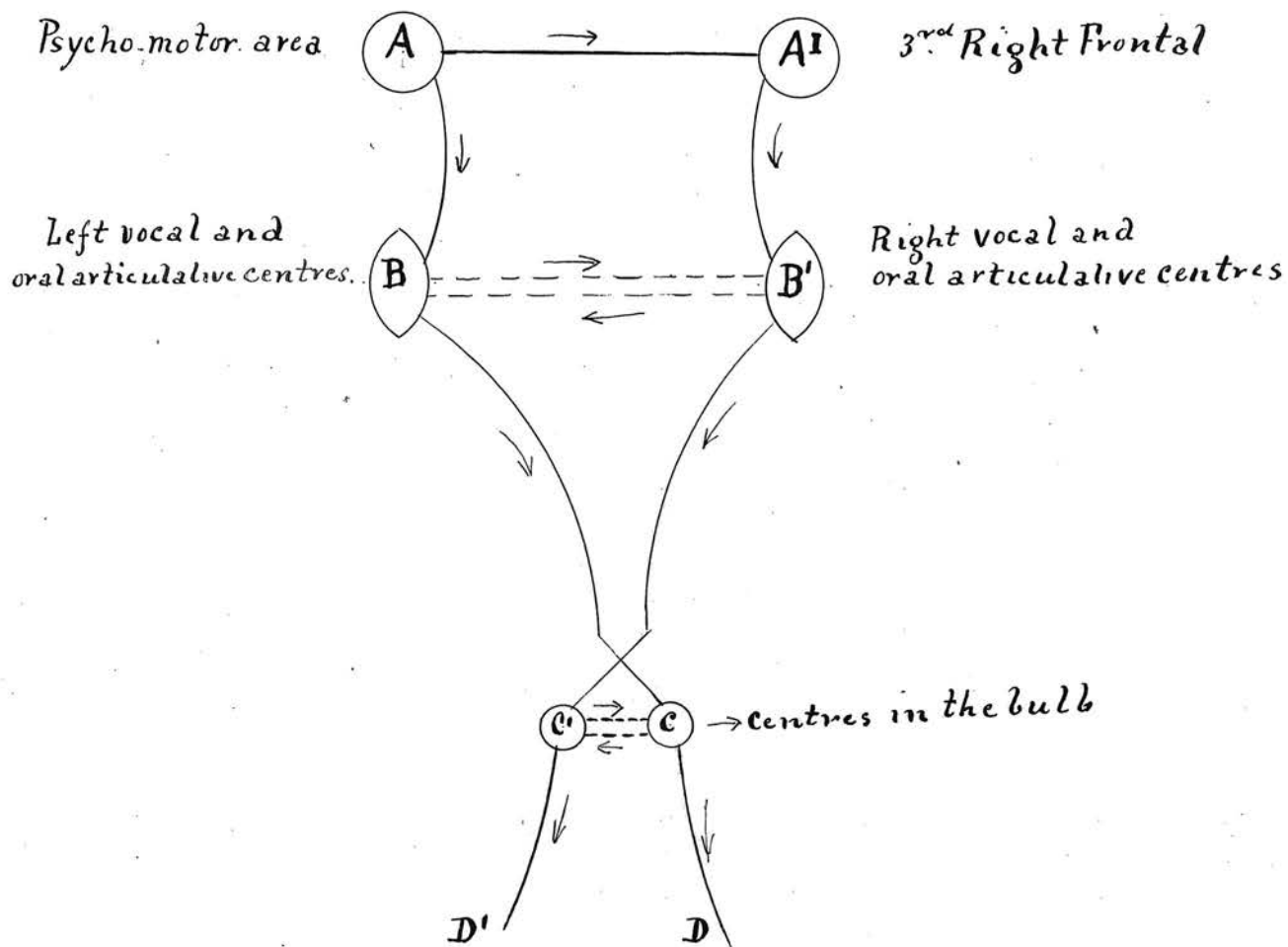


Fig. ix.

. For description see text.

of the speech tract from left to right hemisphere within the brain itself so that the left internal capsule does not contain the whole bundle of these fibres. Fig. IX. is a diagrammatic representation of the course of the fibres of the Motor spoken speech tract.

A is the psycho-motor speech area on the left side.

A' is the corresponding part on the right side.

B The vocal and oral articulative centres on the left side.

B' The corresponding centres on the right side.

C & G, The centres in the bulb.

D, D, The nerves to the vocal and oral articulative muscles.

In speaking, the production route, I suppose to be

A B C D and A' B' C' D'.

If A is destroyed complete motor aphasia is produced

If lesion cuts off A from A' and from B, complete infrapictorial or sub-cortical motor aphasia is produced.

If A is cut off from B alone, or from A' alone, B and A' remaining intact, probably no symptoms or only temporary ones would be produced owing to the commissural fibres between B and B',



Fibres for the tongue
and mouth

Fibres for the wrist,
fingers and thumb

Fibres for adduction of
the vocal cords.

HORIZONTAL SECTION (FLECHSIG).
Through the Internal Capsule and Basal Ganglia.

Fig X

but no case of this has been recorded, the area is so limited. If B is destroyed or B C or if B' is destroyed on B'C' dysarthria results, If B C and B'C' are both destroyed anarthria and pseudo bulbar paralysis result.

From the vocal and oral articulative centres the outgoing fibres pass in the centrum ovale over the lenticular nucleus to the internal capsule, and Horsley and Beevor (32) have shown that the fibres for the oral articulative mechanism are situated at the knee of the internal capsule, whilst Horsley and Semon (33) have shown that the fibres for adduction of the vocal cords are situated in almost exactly the middle of the posterior limb of the capsule. See Fig. X.

The fibres do not form any connections with the corpus striatum as was formerly supposed. They pass with the other motor fibres into the crus cerebri where the motor tract occupies the middle third of the crusta. In the pons the vocal and oral articulative fibres decussate and enter motor nuclei in the floor of the 4th ventricle from which arise the cranial nerves which supply the muscles of the larynx, the throat, the tongue, mouth, lips, and cheeks. Lesion of these fibres on one side in any part of their

course above the bulb produces the same symptoms as destruction of the vocal and oral articulative mechanism centre itself as in case I. viz, no change in the adductor mechanism, but dysarthria in the oral articulative. Lesion of these fibres on both sides produces the same symptoms as lesion of the vocal and oral articulative centres on both sides viz- Anarthria, and the symptoms of bulbar paralysis a form of disease which has been called by French authors pseudo-bulbar paralysis. Below the motor ganglia in the bulb, lesion of the nerves produces of course paralysis in the muscles they supply.

The conclusions we are now in a position to draw from a consideration of the spoken speech production route are the following;-

1st. That the propositionizing or psycho-motor speech centre is in the foot of the 3rd left frontal.

2nd. That the vocal and oral articulative centres are immediately behind it in the lower part of the ascending frontal and the lower part of the ascending parietal convolutions on both sides.

3. That whilst the vocal or phonation mechan-

mechanism is completely bilaterally represented, the oral articulative mechanism is not so completely bilaterally represented in the hemispheres.

4. That the psycho-motor centre is connected directly with the vocal and oral articulative centres on the left side and either directly or more probably indirectly through the less active psycho-motor speech area on the right side by means of connecting or commissural fibres with the vocal and oral articulative centres on the right side.

5. That lesion of the oral articulative centres on either side or fibres from them down to the bulb only produces difficulty in articulation, dysarthria, and not true aphasia, whereas a lesion of the psycho-motor centre or of the fibres coming from it connecting it both to the vocal and oral articulative centres on the same & opposite sides produces complete motor aphasia. Later on it will be shown, however, that the symptoms of a lesion of the psycho-motor centre itself, cortical motor aphasia, differ from the symptoms of a lesion of the fibres connecting

this centre with the vocal and oral articulative centres, (subcortical or infrapictorial motor aphasia).

IV. GRAPHIC PRODUCTION ROUTE.

Let me now shortly indicate the other speech production route viz- the route for writing. In writing the right hand is used by most people and the centre is therefore usually situated in the left hemisphere. Ferrier localises the centre for the hand across the middle of the ascending frontal and ascending parietal convolutions opposite the posterior extremity of the 2nd frontal. In accordance with Broadbent's law the hand is unilaterally represented in the hemispheres, stimulation of the centre in one hemisphere producing only movements in the opposite hand. There is reason to believe that whilst the right hand only is used for writing we can to a certain extent trace letters and form words with the left hand. This I believe in most cases is merely an act of tracing or copying, because the person who is using the left hand even although there is no copy before him raises the visual picture memory of the letters in his cerebrum. It is I believe the same process that is gone through

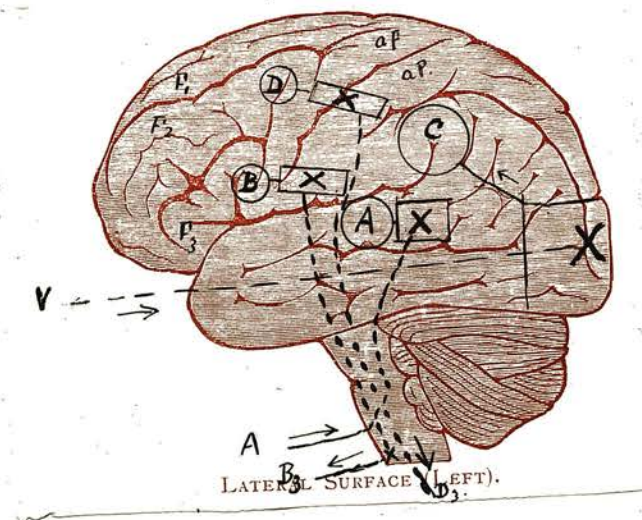
where we try to substitute the left* hand for the right in an action for which the right hand has become specialised, the action is not done neatly and promptly and as it were automatically, but rather by imitating each individual movement of the right hand. This is done either from imitating directly the movements of the right hand or by raising up in memory the picture of its former movements. Later on I shall give reasons for supposing that analogous to the psycho-motor speech centre in the 3rd left frontal there is a psycho-motor writing centre which does all the cell grouping for regulating the movements of the hand, and fingers so that the proper letter is written. This subject, however, will be best considered along with word-blindness and agraphia and I shall only say here that some evidence has been produced in favour of this centre being situated in the posterior part of the 2nd left frontal convolution in right handed persons.

From the motor centre for the hand the fibres pass to the internal capsule where they occupy an area in the posterior limb a little behind the knee whence they pass down with the other motor fibres in the motor tract to the spinal cord, crossing to the opposite side at the decussation of the pyramids and need not further be considered here.

CONCLUSIONS AS TO THE RECEPTION & PRODUCTION,
SPEECH ROUTES.

We have now considered the two routes for the reception of speech and the two routes for the production of speech, and we have been able to localise the terminations and origins of these routes in the cerebral cortex.

We have found that the hearing centres are in both hemispheres in the posterior half of the 1st and 2nd temporal convolutions, and that the visual centres are in both hemispheres in the occipital lobes in the neighbourhood of the calcarine fissure, that analogous to these primary receptive centres - or that part of the cortex where the incoming nerve fibres reach the nerve cells ~~At first,~~ we have on the motor side two areas where the nerve fibres leave the nerve cells in the cortex viz- the lower part of the ascending frontal and ascending parietal convolutions in both hemispheres the beginning of the spoken speech tract, and the middle of the ascending frontal and ascending parietal convolutions in the left hemisphere (owing to the right hand only being usually used for writing) the beginning of the writing speech tract. These areas have been indicated in Fig XI by a X.



- A: Auditory Word centre
- B: Psycho-motor Speech Centre
- C. Visual Word centre.
- D. Graphic Centre.

Fig. XI.

We have seen also that on the production side there is a higher or specialized centre the psycho-motor speech centre situated in the left hemisphere only, in the foot of the 3rd frontal and that there, also probably is a higher or specialized psycho-motor writing centre in the posterior part of the 2nd frontal.

Analogous to these, many cases have proved, that on the receptive side also there are two higher or specialized centres, one, the word hearing centre situated in the same region as, but more limited in area than, the hearing centre and on one (the left) side only, and the other, the word seeing centre situated in the angular gyrus and posterior part of the supra marginal convolution on one (the left) side, only. We shall find evidence of these facts later when we consider the various forms of aphasia.

I have indicated these four specialized centres by an O in the diagram Fig XI and connected them with their lower centres and indicated the course of the speech routes on the receptive side up to these areas and on the production side from these areas.

To complete our consideration of the different routes and to show how these centres are connected it will be best to enter into a consideration of lesions

of these different areas, and the connecting fibres between them,- lesions which give rise to the various forms of aphasia. ~~Before entering on~~

DEXTRAL PRE-EMINENCE.

Before entering on ^{aphasia} ~~this subject~~, however, it

* will be well to say a few words as to why the specialized speech centres are situated in the left hemisphere. Various explanations of this have been suggested. Wyllie (20 page 248) agrees with the explanation first suggested by Dr Moxon (41) that if it is sufficient for the purposes of speech that the speech memories be only stored on one side of the brain then it would be more economical for nature to train one side only instead of both." It has been suggested that the left side of the brain is used in right handed persons because the right hand is used in writing language, and therefore the centre for writing is situated on the left side of the brain, and that if the centres are to be localised to one side it is better for them from an economical point of view to be on the left side. Such a theory, however, besides not explaining why the right hand is used for writing and other actions in preference to the left is

defective also from a physiological point of view because the writing centre is one of the very last to acquire its special function in the production of speech. The child learns to speak before it learns to write and such speech is stored on the left side. The explanation of why the left side of the brain is used in writing as in speaking must be sought for in a consideration of the whole subject of dextral pre-eminence. Many explanations have been offered as to why the right hand takes the foremost place and why either hand is not used indiscriminately. It has been found that not only are the majority of men right handed but they also show a preference for the right leg.

Ogle (42) found that not only do the right limbs take a pre-eminent place in man but that the same dextral pre-eminence is found in some of the lower animals notably in monkeys and parrots. After carefully considering most of the theories as to dextral pre-eminence he arrives at the conclusion that the cause of it is to be found in structural anatomy. The left hemisphere is found to be larger and more convoluted, the left carotid and left vertebral arteries are larger and he thought he found the explanation

of this in the fact that the left carotid came off from the arch of the aorta directly and more in a line with the current of the blood in the aorta, whereas the right carotid is a branch of the innominate which latter vessel at its origin from the aorta is not so directly in a line with, and would offer more resistance to the blood current.

This explanation is certainly very ingenious but it has yet to be proved whether it is the correct one. All that we know at present is that the left side of the brain takes the lead in the large majority of the human race and that it very decidedly does so in the reception and production of speech. It has been found, however, that most left handed persons have their speech centres situated in the right hemisphere and this in spite of the fact that many of them use the right hand for writing showing that writing has not such a decided determining influence as some suppose. This has been found in many cases of left hemiplegia with aphasia where post mortems have taken place. The very few exceptional cases which have been published would probably be found if every circumstance were known to be not really exceptions at all.

IV. MECHANISM OF SPEECH AS SHOWN BY ITSDISORGANISATION.

Having localised the speech reception and the speech production centres to special areas in the cerebral cortex, I shall now proceed by a study of cases of aphasia to show the mechanism by which speech and thought are received, stored up and produced by the brain.

Since Broca's time our knowledge of speech mechanism has gradually been added to. Broca described one form of aphasia, a form which has since borne the name of Broca's type or motor aphasia. James Russell (45) in 1864 was the first to show that besides the form of motor aphasia described by Broca there was another form where there was loss of the memory of words. Ogle (46) for the latter form gave the name of amnemonic and for the former ataxic. Amnesic was soon, however, substituted for amnemonic and amnesia or loss of the memory of words is still much used in connection with aphasia.

Wernicke (13) divided the different forms of aphasia into three varieties; 1st Motor aphasia; 2nd sensory aphasia; and 3rd a form where the conduction fibres between the sensory area and the motor area or

Broca's convolution were involved in a lesion. This form he called *Leitungsaphasie*. Wernicke also localised his sensory form to the temporal lobe and the conduction form probably to the Island of Reil.

Word-blindness was first described by Kussmaul (15) in 1877, but it was not till later that the lesion was localised. Agraphia was noticed in many aphasic patients and was believed to be a symptom of all aphasias but in recent years the tendency of clinical observers has been towards recognising it as a special variety of aphasia, although perhaps the greatest authority on it and word-blindness, Déjerine (17) - has shown that although it is a prominent symptom in the other forms of aphasia the cases are very few if there are any where it alone is the only symptom.

Clinical observation has therefore shown that there are five chief forms of aphasia and four of these forms are produced by a lesion or disorganisation of the four centres which we have already from a study of speech in the child theoretically found to exist, and from physiological, experimental and pathological evidence have localised in special parts of the left cerebral cortex.

These primary varieties of Aphasia are the

following;-

1. Auditory aphasia or word deafness.
2. Visual aphasia or word blindness.
3. Motor aphasia or aphemia or the blotting out of the motor images of words.
4. Agraphia or the blotting out of the motor graphic images of words.
5. Conduction aphasia or lesion of the fibres connecting the auditory and visual to the motor and graphic centres.

The two first will be seen to be sensory and consist in a lesion of the cortical regions where the receptive speech routes terminate, & the next two are motor and are due to lesion of the cortical regions at the commencement of the two production speech routes. Auditory aphasia or word deafness is thus due to lesion in the upper part of the temporo sphenoidal convolution, whilst word blindness is due to lesion in the supra marginal convolution and angular gyrus. Aphemia to lesion in the psycho motor centre or foot of the left 3rd frontal and typical agraphia to lesion in the posterior part of the 2nd left frontal; whilst conduction aphasia cases are usually due to lesions in the neighbourhood of the Island of Reil and floor of the Sylvian

fissure.

Lesions limited, however, to each of these areas are very rare and in most cases we have more than one of these areas involved. And besides the fibres connecting these areas are also often involved so that there are great varieties in the symptoms produced even in cases where the chief lesions are in the same region. From a careful study of the mechanism of speech, however, it is theoretically necessary to add another and higher centre than these four viz.- the centre where the intelligence is called into action or where the concepts are elaborated, which has gone by various names, by various authors but for which perhaps the best name is the "Ideational" centre or centres. Clinical evidence goes a long way to prove that there is such a centre or perhaps more than one centre because some symptoms shown by some aphasic patients are only explicable on such a hypothesis. Going back to our consideration of the acquiring of speech by the child it will be seen that this centre is necessary if the child is to have an intelligent knowledge of the auditory or visual sensory word impressions received as well as an intelligent volitional control over the production speech centres. Completing therefore the

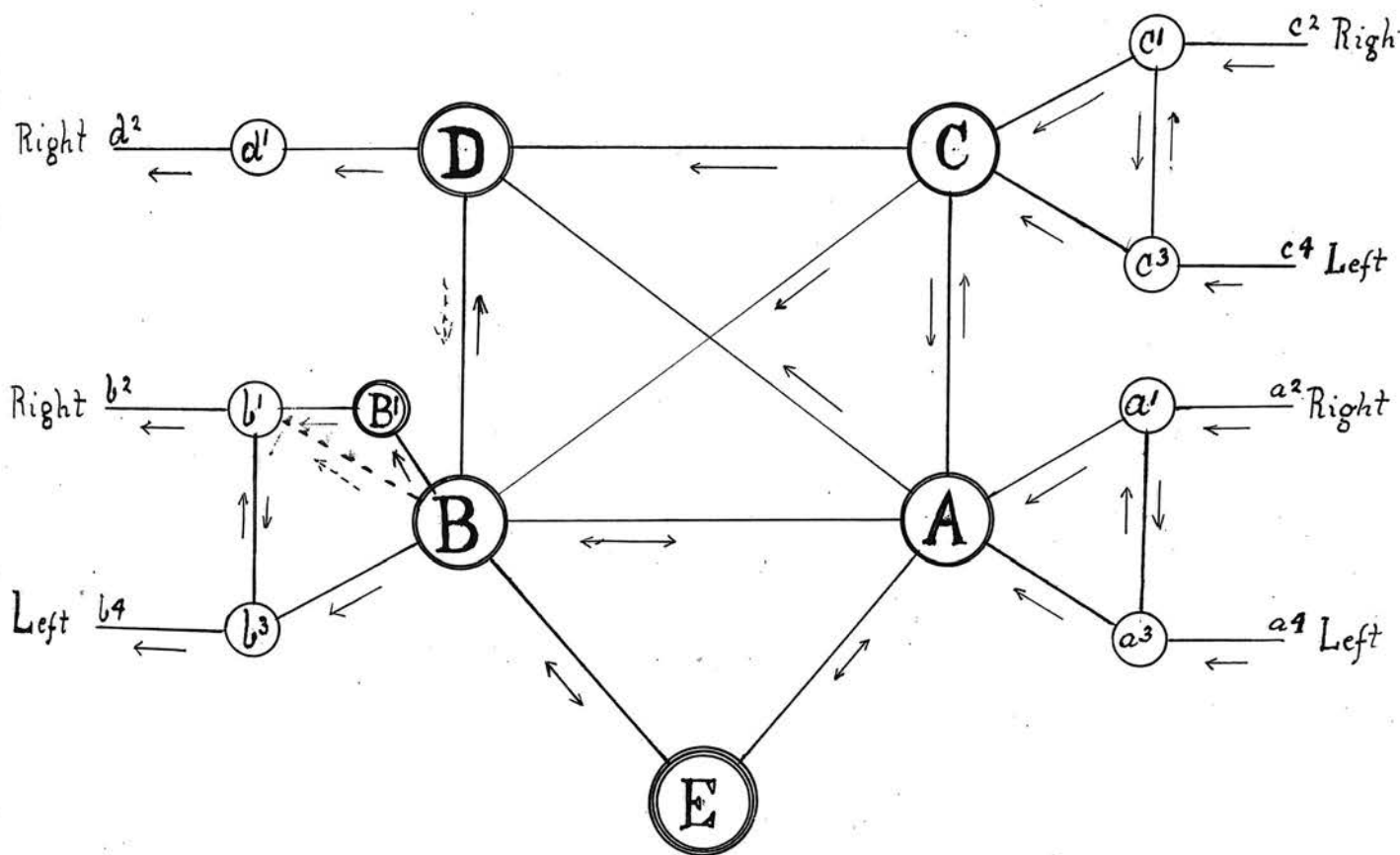


Fig. XII.

schema which I have already drawn out in a previous part of this paper (see Fig. XII.)

A is the auditory word centre, receiving auditory word impressions from a1, the right auditory centre which again is connected with a1, a2, the right auditory nerve, and from a3 the left auditory centre connected with a3, a4 the left auditory nerve. a1 and a3 are probably joined by commissural fibres.

C is the visual word centre, receiving visual word impressions from c, the right visual centre and c2, c1 the right optic tract and from c3 the left visual centre and c4, c3 the left optic tract. c1 and c3 are probably joined by commissural fibres.

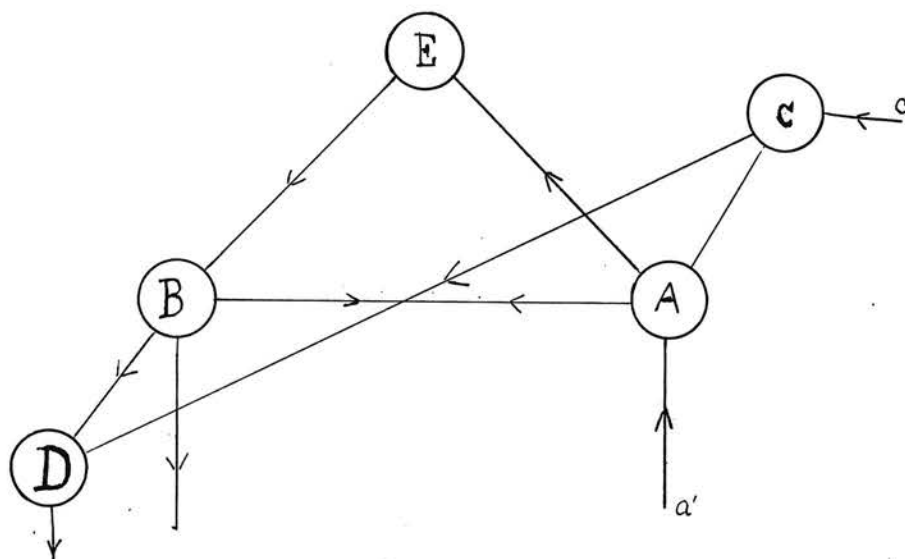
B is the psycho motor speech centre connected with the executory motor centre b3 and motor fibres b3 b4, passing to internal capsule from vocal and oral articulative centres on left side; and connected with b1 - ^tthe executory motor centres on the right side through B' the psycho motor centre; and b1, b2 the motor fibres passing to internal capsule from the executory motor centres on the right side.

b1 and b3 the executory motor centres are probably also joined by commissural fibres. D is the psycho motor graphic centre connected with d1 the

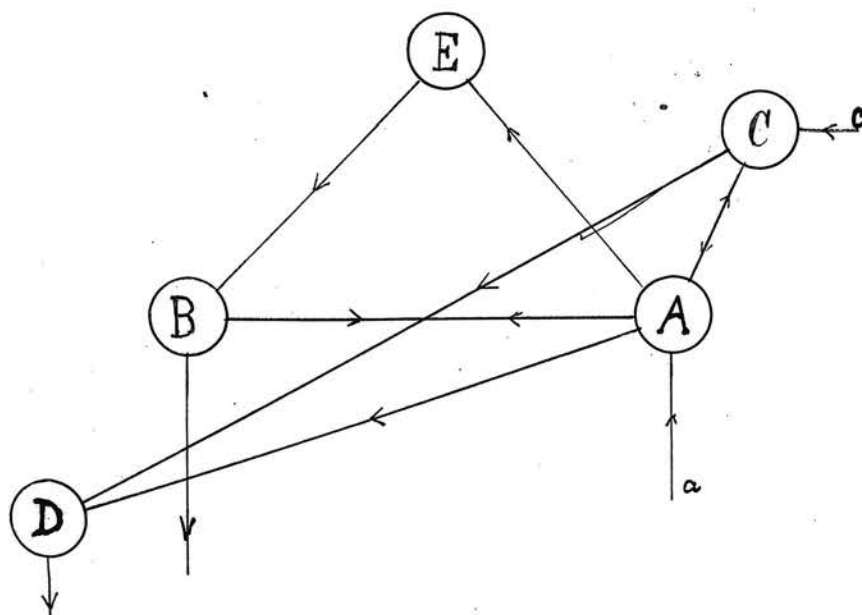
centre for the movements of the hand and d1 d2 the fibres passing from it to internal capsule.

E is the "ideational" concept or "Elaborating" centre and I have connected it with A, the auditory word centre, and B, the psycho motor speech centre, and indirectly through these two with C, the visual word centre, and D, the graphic motor centre, but probably it is directly connected with these centres although these direct connections are not the ones usually used as for instance in ordinary intelligent reading in silence probably in most cases an internal silent articulation of the words as probably also an internal recalling of the sound of the word takes place, showing that if the process does not take place through these centres then by constant association the motor and auditory images are revived by a process of radiation to those centres.

The actual arrangement, however, may be even more complicated than this schema shows and it is necessary to have these centres connected in these various ways in order to produce a schema that will be in accord with all the clinical facts. Many of these schemata have been produced much similar in their principal parts but differing in some of the connections according to the opinion of the authors as to



I.



II

After Lichtheim

Fig. XIII.

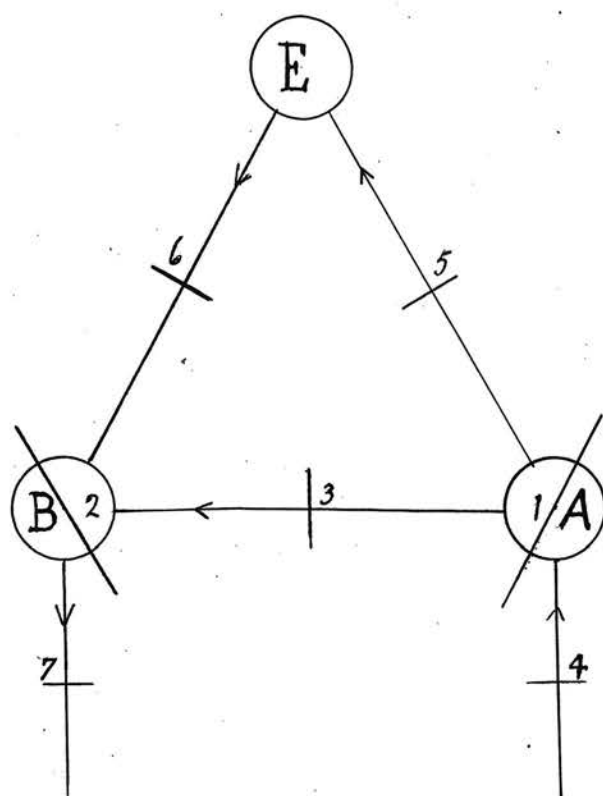
the internal mechanism of speech.

Lichtheim's schema is perhaps better known than any other and is generally accepted as being if not entirely, nearly correct. He really produced two drawings differing in one small detail viz.- in the route of the volitional writing, as will be seen from the two drawings in Fig. XIII.

In the one the route for volitional writing passed from the intellectual centres to Broca's centre and thence to the graphic centre; in the other the route passed through Broca's centre thence to the auditory centre and on to the graphic. In the schema which I have sketched and which agrees in this detail with that of Mills (28) both of these views are adopted.

It will be seen that if a variety of aphasia could be produced by lesion either of any or any number of these centres or connecting fibres, the varieties of aphasia would be very numerous indeed. Practically it has been found that many of these possible varieties are produced clinically because it is possible anatomically to produce them by a pathological process.

Lichtheim taking only the Auditory and the motor centres along with the intellectual centres and putting aside the visual and graphic centres concludes



- 1 = Pictorial Auditory
- 2 = Pictorial Motor
- 3 : Inter-pictorial auditory-motor
- 4 : Infra-pictorial auditory
- 5 : Supra-pictorial auditory
- 6 : Supra-pictorial motor
- 7 : Infra-pictorial motor

Fig. XIV.

that seven different forms of aphasia can be produced by lesions, and that these seven varieties actually occur.

2. The accompanying drawing Fig.XIV. taken from Lichtheim's paper shows the varieties, the cross lines being in the position where the lesions are supposed to take place. Wyllie (20) ~~admits~~^{is} ~~ing~~ that 1 2 and 3 have been distinctly established as varieties that do occur and that Lichtheim's cases go a long way to show that 4.5.6. & 7 also may occur. Wernicke who had adopted Lichtheim's view gave these different varieties of aphasia the following names, but Wyllie suggests that the word Pictorial should be substituted for Cortical in Wernicke's classification. The suggestion is a good one and is not nearly so confusing. These varieties are, the first nomenclature being Wernicke's, the second Wyllie's.

1. Sensory Cortical or Pictorial Auditory.
2. Motor Cortical " Pictorial Motor.
3. Conductive or Leitungsaphasia or Inter pictorial.
4. Subcortical Sensory or Infra pictorial auditory.
5. Transcortical Sensory or Supra pictorial auditory.

6. Transcortical Motor or Supra pictorial motor.

7. Subcortical Motor or Infra pictorial Motor.

Probably a better term for 3 instead of simply Inter pictorial would be inter pictorial-auditory-motor.

It will be seen that if written speech is treated in the same way it is quite possible putting aside spoken speech in the meantime altogether, to theoretically produce seven forms of lesion here also and Wernicke (47) has drawn out a schema and has given names to each of these varieties. I have applied the principle of Wyllie's nomenclature to these different forms. They are.-

1. Cortical Alexia or Pictorial visual aphasia
2. Cortical agraphia. Pictorial Graphic.
3. Conduction agraphia. (Inter pictorial
(visual-graphic))
4. Subcortical alexia. Infra pictorial
visual.
5. Transcortical Alexia. Supra pictorial
visual.
6. Transcortical Agraphia. Supra pictorial
graphic.
7. Subcortical Agraphia. Infra pictorial
graphic.

It will be seen later on that whilst Déjerine and others have proved that 1 and 4 do exist and that there is some evidence to show that 2 also does occur the others are highly problematical as separate lesions

- SYMPTOMS OF CASES OF APHASIA -

Leaving this in the meantime, however, let us now consider what are the symptoms that may be produced theoretically by a disturbance of any part of the central speech mechanism. Looking at our Schema and considering that any of the centres or connections may be destroyed in a case of aphasia the answers to the following questions will make clear what form of aphasia the patient is suffering from and where the lesion probably is.

They embrace practically all the main questions that have to be answered in the investigation of an aphasic case although there are several subsidiary ones, which are dependant on the answer to some of these the principal ones. These questions are;-

1. Can the patient hear sounds of any kind.
2. Can the patient hear words.
3. Can " understand words spoken.
4. Can " see objects of any kind.
5. Can " see words or (can he read words silently)

6. Can the patient understand words written
(can he read intelligently)
7. Can the patient speak voluntarily.
8. Can " repeat words spoken.
9. Can the patient speak words read that is
can he read aloud.
10. Can the patient write voluntarily.
11. Can " write to dictation.
12. Can " copy.

One other question although dependant on the answer to 7,8 & 9 is of so much importance that it will be as well to mention it here viz- If he can't speak voluntarily, repeat what he hears or read aloud, does he know how the word is to be articulated, in other words how many syllables it contains? That is answered by his breathing or pressing the hand once for every syllable the word contains. (L'expérience Proust-Lichtheim.)

These questions as will be seen are arranged in the order in which they would be put by an investigator who was studying the mechanism of speech in the way in which we have approached it, viz- by studying first, the receptive routes and then the productive routes.

I shall take up each of these questions

separately and shall show the significance of the answers to each of them. In doing so I shall refer to the Schema which I have previously drawn out. See Fig.XII.

1. Can the patient hear sounds of any kind?

It is necessary to get an answer to this in order to find out whether sounds reach his cerebral hemispheres at all. If he hears sounds then one or both ears and one or both auditory nerves and hearing centres must be intact a2, a1 & a4, a3. As previously ascertained the auditory nerves are bilaterally represented in the hemispheres so that if the patient is deaf to sounds the lesion must be double and either both ears a2 a4 both auditory nerves a2 a1 & a4 a3 and tracts or both acoustic centres a1 & a2 in the 1st and 2nd temporo sphenoidal convolutions must be affected. A deaf mute is such a case, and I have made reference to several other such cases, of double central lesion when I was considering the localisation of the auditory centres. Such central double lesions must be rare but disorganisation of the organ of hearing in both ears is not quite so rare. Lesion of both auditory nerves and tracts is probably the rarest of all these lesions. It will be convenient to take the next two

questions together.

2. Can the patient hear words? and

3. Can the patient understand words spoken?

Having ascertained that he can hear sounds, it is next necessary to ascertain whether he can hear words. In most cases of course this is quite easily ascertained by simply putting questions to the patient. The best way is to ask the patient to do something or other, such as to touch the nose, ear, etc., but there are theoretically some cases who could not do this and still the auditory word centre would be intact because such a question implies that the person understands the word heard. By looking at the schema it will be seen that it implies that A E is intact. It is possible, however, for A E to be interrupted (Supra pictorial auditory aphasia) and A remain intact. If the lesion was limited to A E then the patient would be able to repeat without understanding words or to write to dictation without understanding the words, such a case has been described by Lichtheim (44 page 454) but probably it is the only one on record so that practically it is easy making out whether the patient hears or not. If he can hear sounds and not words then the lesion is in one of two places 1st a subcortical or infrapictor-

infrapictorial auditory aphasia cutting off both a1 & a3 from A. Such a case must be very rare because as we have already found both the word hearing and the general acoustic centre are in the same region although cases have shown that the word hearing centre is more limited in area and probably only in the posterior third of the 1st temporo sphenoidal convolution. A case of this lesion has been described by Lichtheim (44. Case IV page 461.)

2nd A Cortical or Pictorial Auditory aphasia
destroying the word hearing centre A. Such cases are not uncommon although they are usually not entirely limited to this region. Later on I shall give an account of several such cases which I have personally observed. It may be noted here also that the patient may be able to hear words and not music and to hear music and not words. This point will also be illustrated in one of my cases.

4. Can the patient see objects of any kind?

If he cannot then there is in all probability a double lesion or at all events a lesion bilateral in its results. A single lesion destroying entirely the optic chiasma or both optic nerves or optic tracts would of course produce the same results as double

lesions affecting both eyes, both optic nerves, optic tracts both quadrageminal bodies, both radiations of Gratiolet or both visual centres in the occipital lobes c2, cI & c4, c3. A lesion affecting one side only above the chiasma would produce homonymous hemianopia. I will have more to say on this subject when I consider word-blindness.

5. Can the patient see words?

If the patient can not see words he will not be able to read and it will be found later on that he will not be able to write. Writing is the test for whether the cause of his word-blindness is subcortical that is infrapictorial cI & c3 or Cortical that is pictorial visual aphasia. C. If the lesion is subcortical cutting off C from the visual centres in both hemispheres cI & c3, the patient whilst not seeing words written or printed is able to write words voluntarily written or printed, and to write to dictation. Whilst if the lesion is cortical or Pictorial in C he can't see words nor can he write voluntarily or to dictation. He can, however, copy words as he would any design because he sees objects and words only as objects and not as words. I will have more to say on this, however, when I consider word-blindness and also on other peculiarities of

some cases where there is blindness for words and not for letters, and in others not for figures. .

6. Can the patient understand words written or printed, that is, can he read intelligently?

The patient may be able to see words and yet not understand them. Such a condition might be ascertained by either writing or printing some request or by both, such as "put out your tongue" and although, if the lesion was limited to the fibres between C & E he might be able to read aloud what you had written he would not understand the meaning of your request. He would also be able to re-write it and not understand it. Such a patient would have Supra pictorial visual aphasia but such a condition must be very rare, as an organic lesion if it does occur, although we all know such a functional condition the result of inattention. Another source of confusion would occur, however, because the patient if he could read aloud would hear what he had read and be able to ascertain the meaning by means of the auditory centre. In the schema the fibres have been drawn through the auditory centre A or through the psycho-motor centre B because it is almost certain that in intelligent reading both the auditory and the articulatory image of the word raised

whether that is done by radiation from the visual C or ideational centre E or because the path of the fibres between C & E passes through either A or B or both. Most of us certainly know that we can read even whole pages without understanding what we have read even although we may be reading aloud. The meaning of what we may be reading, however, is not so apt to escape us if we read aloud as it is if we read in silence, showing that the auditory route is useful in aiding written speech to reach the intelligence centres in reading aloud as well as in silent reading.

7, 8 & 9 are best considered together.

7. Can the patient speak voluntarily?

8. ^{read} ~~read~~ words? and

9. read aloud?

It will of course be necessary to ascertain whether the patient is able to speak and not simply whether he does speak or not. Suppose the patient is not able to speak voluntarily then it is possible theoretically that he might be able to repeat words heard or read aloud. Several such cases have been recorded, notably one by Lichtheim (44 Case II) and one by Hammond (48). These cases are supra-pictorial motor aphasia, and result from a lesion of the fibres

connecting E the ideational with B the psycho-motor centres. Such a patient would not be able to write voluntarily but would be able to write to dictation and to copy. If he is not able to speak voluntarily nor repeat words nor read aloud then the lesion is either in the psycho-motor centre B itself or in the fibres from B to the executory motor centres b3 on the left and bI on the right side. If in the psycho-motor centre B the patient has no knowledge of how the words ought to be articulated, Motor Cortical or Pictorial Motor or Broca's aphasia pure, he would also have agraphia.

If in the fibres from the psycho-motor to the executory motor centres (subcortical or infrapictorial aphasia) he would know how the word should be articulated that is he would know how many syllables it contained, and he would be able to indicate that by breathing or pressing the hand once for each syllable. What French authors call "L'expérience Proust Lichtheim". He would also be able to write voluntarily to dictation and to copy. Again the patient may be able to speak voluntarily, to repeat, to read aloud and to write to dictation all of which acts, however, in a disturbed and imperfect manner. This is due to

a cutting off of the connection between the auditory centre A and the psycho-motor centre B and as the fibres from C to B have probably nearly the same if not actually the same course these fibres also are involved. This form of aphasia is the Conduction Aphasia or Inter-pictorial of Wyllie. A prominent symptom in this form is paraphasia and paragrammia. Although the patient is able to speak and to write he uses many wrong words.

When a patient is not able to articulate words properly but knows how to pronounce them (slurring etc) the lesion is either in the executive motor speech centres or in the nerve tracts in the production route lower down - as in Case I previously described and discussed.

10. Can the patient write voluntarily?

11. Can the patient write to dictation?

12. Can the patient copy?

These also are best considered together. The patient may not be able to write because he cannot use the right hand. He may not be able to write at all. In that case the lesion may be in D the psychomotor graphic centre. It is very doubtful whether any true case of this has occurred, one was recorded by Henschen

but there was also a lesion in the angular gyrus, which might have accounted for the agraphia. The lesion may also be in the fibres connecting D the graphic centre with B the psycho-motor speech centre. This would be the position of the lesion if there was no disturbance of voluntary speech, of repeating words, or of reading aloud. If these also were present, the lesion would be as already considered under voluntary speech. If the lesion is in C the word seeing centre itself or in the connecting fibres between C the word seeing centre and D the graphic centre, whether such fibres pass directly C D or indirectly C B D by B the psycho-motor centre or C A B D by A the auditory centre and B the psycho-motor centre the patient is not able to write voluntarily, nor to dictation, but is able to copy as from a design. The first patient would, however, be word blind and agraphic, the second would not be word blind although he would be agraphic. If the patient is able to write voluntarily and to dictation but not to copy except as a design or tracing the lesion is subcortical to the word seeing centre c1 C, c3 C such a patient is word blind but not agraphic.

Having shown how the answers to each of these twelve questions affects the localisation of the lesion

in aphasic patients it will be better to summarise these results and show what reply to each of these questions would be given by an investigator of each of the seven varieties of aphasia described by Wernicke and Lichtheim and renamed by Wyllie.

Question (1). Can the patient hear sounds of any kind and, (4), Can the patient see objects of any kind may be put aside in this connection because they are simply put in order to ascertain whether it is a case of aphasia we have to deal with at all. The answer is presumed in each case to have been in the affirmative.

The symptoms of 7 varieties are given in Lichtheim's paper, but I shall here take the varieties of aphasia in the order in which we would find them approaching the subject from the receptive side just as we have done in the investigation of each part of our subject.

1. Infra pictorial Auditory aphasia (The Subcortical Sensory of Wernicke) Lesion of a1 A & a3 A in schema.
2. The patient cannot hear words.
3. " cannot understand words spoken.
5. " can see words.

6. The patient can understand words written.
7. " can speak voluntarily.
8. " cannot repeat words heard.
9. " can speak words, read, i.e. He
can read aloud.
10. " can write voluntarily.
11. " cannot write to dictation.
12. " can copy.

5.6.7.9.10.12 are thus answered in the affirmative, 2.3.8 & 11 in the negative.

II. Pictorial Auditory. (Sensory Aphasia of Wernicke) lesion of A in the schema.

2. The patient cannot hear words.
3. " cannot understand words spoken.
5. " can see words. (read in silence)
6. " cannot understand words written
(read intelligently.)
7. " can speak voluntarily.
8. " cannot repeat words spoken.
9. " cannot speak words read, that
is read aloud.
10. " can write voluntarily.
11. " cannot write to dictation.
12. " can copy.

5.7.10 & 12 are thus answered in the affirmative.

2.3.6.8.9. & 11 in the negative.

Such a patient although he can both speak and write voluntarily usually shows great disturbances of spoken speech and writing. He usually uses a great many wrong words and wrong syllables both in speaking and writing that is he has paraphasia and paragraphia. The only power that remains undisturbed is the faculty of copying words.

III. Supra pictorial Auditory (Transcortical

Sensory of Wernicke) lesion in A E.

2. The patient can hear words.
3. " cannot understand words spoken.
5. " can see words.
6. " cannot understand written words
7. " can speak voluntarily.
8. " can repeat words spoken.
9. " can read aloud.
10. " can write voluntarily.
11. " can write to dictation.
12. " can copy.

2.5.7.8.9.10.11 & 12 are answered in the affirmative.

3 & 6 in the negative, but as volitional

speaking and volitional writing are done directly from the Ideational centre E and the latter being cut off from the auditory centre there is paraphasia and paragraphia. Although the patient can repeat, read aloud and write to dictation this is done mechanically and not intelligently. It must therefore be done at once, because it cannot reach the intellectual memory. A long phrase therefore cannot be repeated nor written to dictation.

IV. Inter pictorial Auditory-motor (Inter

pictorial of Wyllie, conduction or Leitungs-
aphasie of Wernicke) lesion in A B.

2. The patient can hear words.
3. " can understand words spoken.
5. " can see words (read in silence)
6. " can understand written words
(read intelligently)
7. " can speak voluntarily.
8. " can repeat words.
9. " can read aloud.
10. " can write voluntarily.
11. " can write to dictation.
12. " can copy words.

2.3.5.6.7.8.9.10.11.12 all the questions in fact are

thus answered in the affirmative but there are disturbances in the performance of several of the acts to which these questions refer. 2.3.5.6. and 12 are perfectly preserved whilst there is considerable paraphasia and paragraphia in 7, voluntary language and 10 voluntary writing for the reason stated previously that whilst the direct path from E the Ideational to B the psycho-motor centre is intact that through the word hearing centre A to B is interrupted. There is also paraphasia and paragraphia in 8 repeating words. 9 reading aloud and 11 writing to dictation because instead of these acts being performed directly through the path A B the path used is through A to E the intellectual centre and thence by the voluntary path EB. It will thus be seen how essential the auditory word centre is in the production of correct voluntary language. The ideational centre seems to make use of the auditory word centre in the reviving of the correct word.

V. Supra pictorial motor aphasia (Wyllie) (The transcortical motor of Wernicke) lesion in E B.

2. The patient can hear words.
3. " can understand words.
5. " can see words.

6. The patient can understand words written.
7. " cannot speak voluntarily.
8. " can repeat words.
9. " can read aloud.
10. " cannot write voluntarily.
11. " can write to dictation.
12. " can copy.

2.3.5.6.8.9.11 & 12 are thus answered in the affirmative whilst 7 & 10 are answered in the negative. It seems to me that whilst in the interictorial form (Leitungsaphasie E A B interrupted with the path E B intact, voluntary speech and writing show paraphasia and paragraphia whilst in this form E B interrupted and E A B intact both voluntary speech and writing are lost, the main course of the voluntary speech path is direct from E to B. Nevertheless as we have already seen it seems to be necessary to have the path from E through the auditory centre A and on to B also intact in order to have speech and writing without paraphasia or paragraphia.

VI. Motor Aphasia, Pictorial-Motor Aphasia.

(Wyllie). Cortical Motor Aphasia (Wernicke).

Broca's type of Aphasia. Lesion of B.

2. The patient can hear words.

3. The patient can understand words spoken.
5. " can see words.
6. " can understand words written.
7. " cannot speak voluntarily.
8. " cannot repeat words.
9. " cannot read aloud.
10. " cannot write voluntarily.
11. " cannot write to dictation.
12. " can copy.

2.3.5.6. & 12 are answered in the affirmative.

7.8.9.10 & 11 in the negative.

These answers show that in most cases the path for writing in all probability passes through the psycho-motor centre B. Another point which has not altogether been settled is whether such a patient is able to read silently and understand what he is reading. According to the schema, he can, but it is very doubtful whether all cases are able to do so or not. In the not very well educated individual probably the faculty of reading in silence and intelligently would be lost, because such persons almost invariably use articulation more or less audibly, or if not at all audibly then they are conscious of the movements necessary for the articulation of the words. Even most

if not all educated persons are conscious of the same thing as well as of a more or less distinct auditory image of the word. Probably as a person gets more and more proficient at reading in silence such images get less and less distinct but it is very doubtful whether they entirely disappear. Sometimes when a language is acquired almost entirely by reading and not by hearing or speaking as for instance in the acquiring of a foreign language, such articulative images may not be called up on reading foreign words. Some persons have acquired foreign languages in this way and don't recall the articulative image because they have never used articulation in the acquiring of the language. This must apply more distinctly to a language where the sounds and pronunciation of the words are different from the person's native language. The same remarks apply to the faculty of writing silently. The oral articulative images of the letters or syllables are probably always raised in the memory, hence the faculty of writing is almost always affected when the psycho-motor speech centre B is destroyed.

VII. Infra pictorial Motor Aphasia (Wyllie)

Subcortical motor Aphasia (Wernicke). lesion in

Bb3 BbI.

2. The patient can hear words.
3. " can understand words.
5. " can see words (read in silence)
6. " can understand words written.
7. " cannot speak voluntarily.
8. " cannot repeat words spoken.
9. " cannot read aloud.
10. " can write voluntarily.
11. " can write to dictation.
12. " can copy.

2.3.5.6.10.11 & 12 are answered in the affirmative.

7.8. & 9 in the negative.

This therefore differs from Broca's cortical type in that the faculty of writing remains intact. The patient is able to indicate how the words ought to be articulated, he knows the number of syllables the words contain. The faculty of writing is preserved because the psycho-motor speech centre is intact but only cut off from the executory motor centres of its own and the opposite side. Two functional cases of this lesion I shall describe later.

I shall now take the seven varieties of written speech lesions which have been theoretically assumed to exist by Wernicke and shall take them in the same

order, Viz- from the reception to the productive side. As no cases of some of these lesions have yet been described there must necessarily be some uncertainty as to the precise symptoms.

VIII. Infra pictorial visual Aphasia of Wyllie.

Subcortical Alexia of Wernicke. Cécité verbale
pure of Déjerine and French authors. lesion in
cI C. c3 C is one of the best known having been
very carefully observed by Déjerine (17) Sérieux
(18) etc. It is usually accompanied by hemian-
opsia owing to an involvement of the radiations
of Gratiolet as well as the fibres subcortical
to the angular gyrus.

2. The patient can hear words.
3. " can understand words spoken
5. " cannot see words.
6. " cannot understand words written
7. " can speak voluntarily.
8. " can repeat words.
9. " cannot read aloud.
10. " can write voluntarily.
11. " can write to dictation.
12. " can copy (only imperfectly as
in tracing a design)

2, 3, 7, 8, 10, 11, 12 are answered in the affirmative. 5, 6, 9 in the negative.

The difference between this form and the next is that the patient can write when subcortical whereas he cannot do so when a cortical lesion.

IX. Pictorial Visual Aphasia. Cortical Alexia of Wernicke. Cécité Verbale avec Agrophie of Dejerine, etc., (lesion of C.)

2. The patient can hear words.
3. " can understand words spoken.
5. " cannot see words.
6. " cannot understand words written.
7. " can speak voluntarily.
8. " can repeat words.
9. " cannot read aloud.
10. " cannot write voluntarily.
11. " cannot write to dictation.
12. " can copy only imperfectly.

2, 3, 7, 8, answered in the affirmative.

5, 6, 9, 10, 11, and 12 in the negative.

I will have much more to say about these two forms when I consider word-blindness.

X. Supra Pictorial Visual Aphasia. Transcortical Alexia of Wernicke lesion in C E, which probably passes through A and therefore if lesion

between A and E the symptoms would be the same as III Supra Pictorial Auditory Aphasia; and if A was involved the symptoms would be as Pictorial Auditory; but if between C the visual and A the auditory centre the symptoms would be

2. The patient can hear words.
3. " can understand words spoken..
5. " can see words written.
6. " cannot understand words written.
7. " can speak voluntarily.
8. " can repeat words spoken.
9. " probably cannot read aloud.
10. " can write voluntarily.
11. " can write to dictation.
12. " can copy.

2, 3, 5, 7, 8, 10, 11, and 12 answered in the affirmative.

6 and 9 in the negative.

It is a little doubtful as to whether such a case would be able to read aloud or not. It depends on whether the route from the auditory centre C passes directly to B the psychomotor centre or indirectly through the auditory centre, probably the latter ~~route~~ is the correct ~~route~~ because in lesions of the auditory word centre the patient is not able to read aloud. There is also some doubt

as to whether a patient with this lesion could write either voluntarily or to dictation, the answer to this depends on whether the route from C to D is direct or through the auditory centre and also as to whether there is a true graphic centre in D or not.

XI. Inter Pictorial Visual-graphic Aphasia,
Conduction Agraphia of Wernicke lesion in C D.

The course such fibres may take whether direct C to D or indirect C B D or C A B D is not known. In writing most of us are conscious not only of the visual image of the letters we write but also of the auditory and articulatory images, showing that if the route is not through A and B there must be a radiation of nerve influence from C and D to A and B. If the course is direct from C to D.

2. The patient can hear words.
3. " can understand words spoken.
5. " can see words.
6. " can understand written words.
7. " can speak voluntarily.
8. " can repeat words.
9. " can read aloud.
10. " can write voluntarily,
11. " can write to dictation.
12. " can copy.

It will be seen that all these questions are answered in the affirmative although as in inter-pictorial auditory-motor (*Leitungs - aphasia*) there might probably be paragraphia and other writing disturbances. The alternative indirect route through the ideational, auditory, and psychomotor centres would in all probability be used, but as no case has been described it is impossible to say what exactly the symptoms of such a lesion may be.

XII. Supra Pictorial Graphic Aphasia. Trans-cortical Agraphia of Wernicke.

This variety also is highly problematical ^{as an isolated lesion} probably the route from the ideational centre E to the graphic centre is through B the psychomotor speech centre. If the lesion is therefore between E and B the symptoms would be as in Supra pictorial motor aphasia type.V., if between B and D the symptoms would be

2. The patient can hear words.
3. " can understand words spoken.
5. " can see words.
6. " can understand words written.
7. " can speak voluntarily.
8. " can repeat words.
9. " can read aloud.
10. " cannot write voluntarily.
11. " cannot write to dictation.
12. " can copy (as a design).

2, 3, 5, 6, 7, 8, 9, and 12 answered in affirmative. 10 and 11 in the negative. The route used in writing to dictation is probably through B the psychomotor centre as its destruction as in Brocas type of aphasia causes agraphia to everything but copying.

XIII. Pictorial Graphic Aphasia Cortical Agraphia of Wernicke lesion of D the psychomotor graphic centre if such exist. The symptoms would be

2. The patient can hear words.
3. " can understand words spoken.
5. " can see words.
6. " can understand words written.
7. " can speak voluntarily.
8. " can repeat words.
9. " can read aloud.
10. " cannot write voluntarily.
11. " cannot write to dictation.
12. " cannot copy.

2, 3, 5, 6, 7, 8, 9, are answered in the affirmative. 10, 11 and 12 in the negative. This is a pure agraphia such as described by Henschen.

XIV. Infra Pictorial Graphic Aphasia, Sub-
cortical agraphia of Wernicke (Lesion in fibres
 between D and d 1.)

2. The patient can hear words.
3. " can understand words spoken.
5. " can see words.
6. " can understand words written.
7. " can speak voluntarily.
8. " can repeat words.
9. " can read aloud.
10. " cannot write voluntarily.
11. " cannot write to dictation.
12. " cannot copy, except by tracing as a design and only then if the right hand is not paralysed.

2, 3, 5, 6, 7, 8, and 9 are answered in affirmative.
 10, 11, and 12 in the negative.

These results are the same as in the last form but the difference in this form would be that the patient would know how the word and letters ought to be written but would not be able to do it. This form is however highly problematical *as an isolated lesion*.

These results I now put down in a tabular form.

TABLE OF SUMMARY OF RESULTS OF ANSWERS TO THE
12 QUESTIONS IN EACH TYPE OF APHASIA

Type	Affirmative												Negative											
I.	1.	4.	5.	6.	7.	9.	10.	12.					2.	3.	8.	11.								
II.	1.	4.	5.	.	7.		10.	12.					2.	3.	6.	8.	9.	11.						
III.	1.	2.	4.	5.	7.	8.	9.	10.	11.	12.			3.	6.										
IV.	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.												
V.	1.	2.	3.	4.	5.	6.	8.	9.	11.	12.			7.	10.										
VI.	1.	2.	3.	4.	5.	6.				12.			7.	8.	9.	10.	11.							
VII.	1.	2.	3.	4.	5.	6.			10.	11.	12.		7.	8.	9.									
VIII.	1.	2.	3.	4.		7.	8.		10.	11.	12.		5.	6.	9.									
IX.	1.	2.	3.	4.		7.	8.						5.	6.	9.	10.	11.	12.						
X.	1.	2.	3.	4.	5.	7.	8.		10.	11.	12.		6.	9.										
XI.	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.												
XII.	1.	2.	3.	4.	5.	6.	7.	8.	9.		12.					10.	11.							
XIII.	1.	2.	3.	4.	5.	6.	7.	8.	9.							10.	11.	12.						
XIV.	1.	2.	3.	4.	5.	6.	7.	8.	9.							10.	11.	12.						

From these results it will be seen that it is possible to localise every lesion producing a disturbance of speech if that lesion is limited to a particular centre or the connecting fibres between centres. Probably there is not much difference between Type IV Interictorial auditory-motor and XI Interictorial visual-graphic, the chief symptom of both being paraphasia and paragrammia, the latter symptom being perhaps more marked than the former in XI. If lesions are to be accurately localised it will be seen from a study of this table how essential it is that cases should be accurately observed in all their aspects. An answer to each of these 12 questions which I have drawn up if accurately obtained in each case would not only increase our knowledge of ap^hasia cases generally but enable us to localise almost every one. It will be shown later on, how important such localising is in cerebral surgery, and I may repeat here that I look upon aphasia as being quite as much a symptom which ought to be thoroughly studied and understood by the Surgeon as by the Physician, indeed I think its localisation is much more important in surgery than in medicine. A study of the cases of aphasia which have been recorded in Medical literature very soon leads to the conclusion that the accuracy in every detail with which they have been observed is not the distinguishing feature of them. Probably this is

to be accounted for by the fact that our knowledge of aphasia has rapidly grown and widened and no method of accurate observation has been in universal use. To supply this want Wyllie has carefully drawn up a method of case-taking which if universally adopted would very soon be the means of adding materially to a better knowledge of all the central affections of speech.

V. CLINICAL VARIETIES OF APHASIA ILLUSTRATED BY CASES.

To return, however, to our consideration of the different varieties of aphasia although it is possible to have all these different Types and cases of many of them have been recorded practically the greater number of aphasic lesions involve more than one centre or sets of fibres so that the cases are usually more complex than those forms described. This results from the fact that such lesions are most commonly of vascular origin, either a haemorrhage or a blocking of a vessel from a thrombus or an embolus. As such vessels supply more than one cortical area, more than one cortical area is usually involved in the pathological process. Of course sometimes we find a small haemorrhage or an abscess

or cerebral tumour which picks out a small area. In those cases the symptoms may closely resemble one of the types of aphasia described. As a rule however, as I have said, the lesion is not so limited and therefore the combination of symptoms varies in different cases. It has been found as I previously stated that in a large number of cases it is possible to divide aphasic cases into 5 clinical groups which have gradually come to be recognised as the 5 different kinds of aphasia. These five are:-

1. Auditory aphasia, comprising Type I. II. & III
2. Motor aphasia or aphemia comprising Type V. VI. and VII.
3. Visual aphasia comprising Type VIII IX & X.
4. Graphic aphasia comprising Type XII XIII & XIV
5. Conduction aphasia (Leitungsaphasie) comprising Type IV & XI.

The latter group are produced by lesions of the fibres connecting the sensory to the motor side. They perhaps most closely resemble the Auditory group and will be considered immediately after that group. I shall therefore take up in order the consideration of these five groups of aphasic lesion, giving cases of my own observation, illustrating the different groups.

I. AUDITORY APHASIA

- including
1. Infra Pictorial Auditory.
 2. Pictorial Auditory.
 3. Supra Pictorial Auditory.

The following two cases of my own observation are excellent examples of Auditory aphasia.

The first case is one of very great interest not only from the fact that it was almost a pure pictorial auditory and pictorial visual case but also from the fact that it was one of those cases of rapid recovery which have been referred to by Wernicke. The explanation of such recovery has been sought for in the fact that the function of the left auditory word centre is taken up by the right side. Later on I shall give reasons for supposing that such an explanation does not explain some cases such as this, and that the true explanation is to be sought for rather in the theory that there are functional sensory aphasias as well as functional motor ones. Several good examples of the latter I shall also record later.

CASE II.

(Personal observation).

Pictorial Auditory and Pictorial Visual aphasia,
sudden in onset; temporary in duration; quick, almost

immediate recovery; some paraphasia; marked amnesia chiefly of nouns; no motor symptoms; musical faculty affected slightly in a peculiar way.

S.C. aged 18, a domestic servant, was admitted to Leith Hospital on 16th March 1895. Her father died of apoplexy, otherwise there is no history in the family of cerebral disease.

When a child she had measles, and scarlet fever, the latter being followed by an abscess behind the left ear which however, very soon healed up. Since then she had enjoyed good health.

Two months before admission she complained of pain in the left temple, but was able for her work till Monday 11th March. This pain was thought to be neuralgic in nature, and was treated with quinine. On 11th March she felt sick and was unable for work, but the pain in her head was no worse. On the 12th March, it shifted to the nape of the neck, and on the night of the 12th patient was very dizzy when she got up. There was no disorder of speech on the 13th. On the 14th at 2 a.m. she spoke to her fellow servants quite well. Her head was then very painful and she got up and applied vinegar and water to it. At 6 a.m. it was noticed that she could not use the proper words. The words she used were "Snow," "Auntie," "Egg" and "God," but she could

always show where she felt pain, and would say "Sore there" or "Fine." She said she heard a song in her ear, and actually whistled it. It was a song called "Molly Reilly." On Thursday she was seen by a Doctor and ice was applied to her head. She was tried with written words but she could not understand them. She was admitted to Hospital on Friday the 16th March.

Condition on Admission.

She was a bright healthy looking girl with good colour and well nourished. Examination of the circulatory system showed pulse regular and of fair strength. Heart sounds were quite pure and no murmure, no enlargement or other abnormality. The Respiratory, Alimentary and Urinary systems were quite normal.

Nervous system. Ordinary motor and sensory functions as well as the reflexes were normal. Examination of the speech disturbance according to Wyllie's "Method" showed:-

1. Spoken Speech. (a) How received and interpreted? The hearing was good. If any noise was made or word spoken, she at once turned in the direction of the sound.

With regard to interpretation of words? She seemed to understand only those which had frequently

been mentioned to her. Thus she at once put out her tongue when asked, because she had been requested to do so before and was expecting the question. At first when asked to shut her eyes she was unable to understand, subsequently she did so quite readily. When asked to give her hand, she could not understand what was meant, and said "I forget." She was unable to understand such words as nose, ear, etc. When asked to touch her nose, ear, eyes, or chin, she shook her head, evidently was distressed, and rubbed her eyes and looked enquiringly at the nurse who was standing near. She was quite unable to understand such words as "smile", "whistle", "shut your eyes."

(b) How was spoken speech produced? She spoke quite distinctly, there was no slurring of words.

Some common objects were shown to her. A watch she at first called an egg, a pencil a book, and an orange she was unable to name, but when the name was mentioned she smiled and nodded her head. Milk she called "snow." A handkerchief and an umbrella she also called "snow." She recognised her mother, and called her by that name. Almost all other people she called "Auntie" and sometimes common objects were called "auntie." A candlestick she called "God." The candle and the light were called "snow." When asked her own name, she said "Sarah." Asked how

she was, she always was "fine." Have you any pain? She pointed to the left temple and said "sair there." Sometimes she seemed conscious of having used the wrong word, and looked distressed saying "I forget." When the right word was mentioned on these occasions she smiled and nodded her head. Usually, however, she was not conscious of her mistakes. She could not be got to repeat words or phrases.

II. Written speech. (a) How received and interpreted? Sight was good. There was no hemianopsia. She was unable to understand questions in writing, such as "Put out your tongue." She could make out the letters of the alphabet, but could only understand small and common words, such as "to," "so," and "merry." "Knit" she called "fen," "by" she called "to."

(b) How written speech was produced. Her name Sarah she wrote easily. Her surname she wrote with more difficulty and hesitation, but correctly. She was unable to write answers to questions or to dictation, except numerals (for instance 31 was written correctly) She failed to copy correctly words written on paper. If asked to write down any word she usually began it with a "P" for instance, for eighteen she wrote "Pe," for snow she wrote "Pno" for aunty she wrote "Pumrain" and for God she wrote "Pumrase." She understood gesture language well.

She understood the use of some common objects, such as a watch. She was shown one at 7.50, and after examining it closely said "Ten to three." She then pointed out and named in sequence the various hours 1. 2. 3. 4. 5. up to 12.

Progress.

Her temperature which was 101° on the day of admission fell to 99° next day, was 100° on the 18th, and normal on the 19th after which it remained normal. On the 20th it was noted that her aphasic condition was very much the same, except that she now having repeatedly been asked certain questions understood them better. Thus she always at once put out her tongue and shut her eyes when asked to do so. She had more difficulty when asked to touch the ear or the eye. When asked to touch her ear she said "This?" pointing to her eye. Then she pointed to her ear and smiled and nodded. When asked to shut her eyes she did so at once.

31st March. Her condition was much the same as on the 20th March. On the evening of that day a sudden improvement set in. She asked the night nurse for milk. This she had hitherto called "snow." She now had little difficulty with the names of ~~thing~~ things, but was easily excited, and when so, was apt to use the wrong word but was at once conscious

that she had done so. For instance she asked if she might be allowed to get up and sit at the window. This she corrected at once, saying "No, no, I mean the fire." She could read now quite easily.

8th April. She seemed now quite recovered, she had no difficulty at all in expressing her wants. She still complained of pain in the left side of the head. She had no recollection of the precise onset of her illness, and had a very vague idea of the difficulty she had experienced with her speech during her illness.

20th April. She was discharged quite well.

From these notes which were very carefully taken by the House-physician Dr A.W.Cameron, it will be seen that this patient had in addition to Auditory aphasia, more difficulty with written speech both in its reception and production than is usual in a lesion of the auditory word centre pure and simple.

Tabulating the answers to the questions I have already drawn out, the results are the following.

1. The patient could hear sounds.
2. " could not hear words.
3. " could not understand words spoken
4. " could see objects, etc.
5. " could see letters and small words but not long ones.

6. The patient could not understand words written.
 7. " could speak voluntarily but showed paraphasia and difficulty in recalling correct words.
 8. " could not repeat words spoken.
 9. " could not read aloud.
 10. " could not write voluntarily, except a few words and had paraphasia.
 11. " could not write to dictation.
 12. " could not copy.
1. 4. 7. answered in the affirmative.
2. 3. 5. 6. 8. 9. 10. 11. 12 in the negative.

Comparing this with the results of our theoretical study of the types of aphasia we see that there are none of the forms which precisely correspond to this case. It will be seen, however, that if we take the symptoms of Type II Pictorial Auditory Aphasia and add to them the symptoms of Type IX Pictorial Visual Aphasia that we get exactly the results we had in this case. It was thus a very pure case of Sensory Aphasia involving chiefly if not entirely the cortical areas in which are situated the word hearing and probably also the word seeing centres. In all auditory cases there is a difficulty in saying exactly whether the visual word centre is also involved because of the fact previously mentioned that the

auditory word centre has to be intact in order to see words intelligently. Other points to notice in this case are the following.

(1). The very distinct forgetfulness of the names of things, amnesia of nouns, a subject I will have to say more about later.

(2). The distinct retention of knowledge of and production of gesture language.

(3). The retention of the understanding of heard music as well as the power of reproducing a melody by whistling.

(4). The disturbance of the music hearing faculty insomuch as the patient had "hal^lucinations of melody."

(5). It is also to be noted that there was complete agraphia even of the power of copying, probably entirely due to the visual word centre being involved.

(6). She had paraphasia and paragrammia as well as "word intoxication" or the tendency of reproducing over and over again the same letter or word in writing and speaking. It will be seen that the word "snow" was used in answer to all sorts of questions, and that in writing the same letter was apt to appear again and again. This is a very common symptom in auditory and conduction aphasias.

In pure auditory cases the patient does not know when the wrong word is spoken, whereas in pure conduction cases the patient hears the mistaken word, and is annoyed when the wrong word or the same word is spoken again and again..

(7). That the patient could see letters and figures as well as very small words although the power of seeing longer ones was almost entirely abolished.

(8). The temporary nature of the case and the sudden and rapid recovery.

The preceding was a case of recovery of a very pure cortical auditory and visual aphasia. The next is one of 17 years duration, and the diagnosis was verified by a post mortem examination. The case had been observed very carefully at various times during its progress, and notes had been taken. For the notes of the case which had been taken whilst the patient was in Morningside Asylum, I am indebted to the kindness of Dr Clouston, for those taken whilst under my care in Leith Hospital, to Dr Hill Buchan, House-physician. The brain of this case was shown to the Edinburgh Medical-Chirurgical Society in January 1896.

CASE III

(Personal observation).

Auditory Aphasia of 17 years duration. Hemi-
plegia at first, but gradually recovered from; sensory
aphasia symptoms persistent; marked amnesia, chiefly
of nouns; paraphasia; Agraphia; insanity; Post Mortem

Mrs E.S., aged 53, was admitted to Leith Hospital on September 26th 1895, in a semicomatose condition with the following history of her illness.

About three weeks before admission she began to vomit after food and this symptom lasted a week. She had pain in her head, and after the first week had to keep entirely to her bed. She became gradually more stupid and weaker and less able to recognise her friends, and for about a week before her admission she was unable to recognise anyone at all.

The following was her state on admission. She was lying in a stupid semicomatose condition, eyes almost closed, face slightly drawn to left, no motor paralysis in legs and arms. Right plantar reflex almost absent, left rather exaggerated, knee reflexes normal. No atrophy of muscles of either side. Pupils "pin point," slight external strabismus of both eyes; patient occasionally gave a deep sigh and an attempt to utter something. She

could swallow though there was considerable difficulty in the act. Pulse 115; no heart murmur; no respiratory change; bladder was distended, and a large quantity of urine was drawn off which had a specific gravity of 1030 and contained no albumin and no sugar. The following history was got from the friends. Patient is the mother of eight children of whom one died about three months old. This child was born 17 years ago on 8th December, the confinement being easy. Fourteen days after when she had been up and washing clothes she went to bed all right. At 7 a.m. next day her husband found her lying in bed "stiff and quite unconscious." The right arm and leg were paralysed and the face twisted. Patient began to recover about a month after the onset of the illness (the middle of January). At first she could not speak at all but gradually slight improvement appeared, and went on for six or eight months and since then her speech has remained in statu quo. Her condition since then is described by her husband as follows:-

(1). She could speak words but could not converse. Her vocabulary was very small. She could say "yes" or "not" and some expression, such as "Fine laddie," "fine lassie." She was never able to address her friends, even her husband by their names, but would indicate them by some object :

connected with them. If wishing to speak of a friend who was a "butcher" she would nod and say "the beef" similarly if she wanted provisions bought she pointed to sugar, etc. She could not tell the days of the week though she knew them quite well. She indicated her various children by pointing to their respective heights. She would sometimes ask "are you going out?"

II. Understanding of spoken words.

Such a sentence as "I am" she quite understood, but if one said to her "I am going to Mrs Welsh's (her most intimate friend) she did not understand unless some object connected with Mrs Welsh was pointed to. Her understanding of spoken words was very limited. The method of conversing with her on the part of her friends was almost entirely by signs. To indicate her brother, a plumber, they pointed to the gas bracket. Her hearing for sounds was quite acute.

III. How did she understand written speech?

Previous to her illness she was a great reader, but since then she has never been able to read a book, though she frequently tried to. She was, however, able to recognise her husband's and any of her family's names in writing, but if, say the word "pen" was written, she could not recognise it. Any-

thing, however, in butchers' and bakers' books if too heavily charged, she challenged at once, for instance on one occasion a whole loaf had been charged for whilst half a loaf only had been sent, and she corrected it at once. On the other hand an error in calculation she could not correct. (Could not count up). She apparently saw figures, but could not calculate.

IV. Could she write?

She was quite unable to write, not even her own name after her illness. It is noted in the Morningside report that she could copy. At the end of seven or eight months she had regained almost entirely the complete use of her leg and arm, the paresthesia of the leg disappearing first. In July she saw an Edinburgh consultant. She then appeared very stupid and complained of pains in her head, making signs as if to indicate that she had a feeling as if something in her head was going round and round, and pointing to her head she would often say "noise." Her removal to Morningside Asylum was advised and accordingly she was taken there. The following notes were taken at that time in Morningside Asylum.

Her Medical certificate stated that her look was peculiar, fixed and vacant. When asked questions about her condition she spoke unintelligible

nonsense. Appearance indicates chronic organic disease of brain as cause of her insanity. She lost power of one side as result of paralytic shock about 9 months ago, and since then has never been of sound mind or able to attend to her duties or behave rationally. Her sister-in-law said that since the shock she had never been of sound mind, been sleepless, refused food, and was totally unable to attend to her duties or children. Her state on admission was. She was depressed, showed considerable enfeeblement, memory much impaired, coherent at times, generally not; Answered questions generally in an absurd rambling way. Pupils unequal, left larger than right, sluggish. Had partial aphasia. Her sensory nervous system was dulled. Tongue tremulous and pointed to left side. Disease mania. Did not improve and after a few weeks was transferred to Larbert Institution, where she remained seven or eight months. On leaving Larbert, she was, her husband says, mentally all right, complaining only of dizziness when she stooped.

About five years ago in July 1890, whilst out walking she took drink and became confused and excited. When she took drink, even one glass, she became stupid, it was impossible to understand what she said or get her to understand what was said to

her. The result was that she was found wandering by the police, and was again certified as insane and sent to Morningside Asylum, her medical certificate stating "that she had obstinate taciturnity, quite unable to give any account of herself, does not know her name, talks incoherently."

The following notes were taken in the Asylum.

She is slightly depressed, complains of pain in head. There is slight enfeeblement, confused, memory good. Is incoherent. Can only answer some questions, no delusions. Appearance is not unintelligible^{ent} looking.

Nervous system. (Motor) Forehead, left wrinkles slightly higher than right, left lower eyelid less full. Left upper lid one deep fold, right several folds, none very deep. Eyes Kalmuck. Right nasal groove shallower than left. Tongue on left side of mouth, points to right. Special senses not paralysed on any side. Pain on tapping head, especially on left parietal region. Arm and knee reflexes more marked on left, tongue shaky. Disease melancholia. Owing to her aphasia, patient could not tell her name and address, so that she was here for a few days under the cognomen "unknown." The excitement passed off in two or three days, and then she became a case of simple aphasia, but a most

interesting one. She suffers from partial amnesic aphasia. She put out her tongue on being asked, but as a rule did not understand what was being said to her. Cannot answer her name. Asked her age said 40 and 3, makes fair simple reflex replies. After several times pressing told the time (20 to 9) Cannot answer what requires thought. Can't write, but copies writing.

She was only a short time in Morningside Asylum and was discharged cured of her mental symptoms. About the month of September, three years ago, she suddenly went, as her husband says, "into fits." During the day she had been in her usual health, and in the evening she complained of pains in the head and went to bed. About 8 p.m. she suddenly became unconscious and went into convulsions. No very full account of this from her friends, but they say that "she worked most on the right side," "that her face went to the right" and "that the right eye winked most." She was half an hour in the first attack. At the end of it she lay unconscious, and "like a stone for quarter of an hour" when a second one, lasting twenty minutes, came on. The convulsions went on with only about minute intervals, till 2 p.m. when she fell asleep and did not awake till mid-day next day. The stupor gradually passed off and in

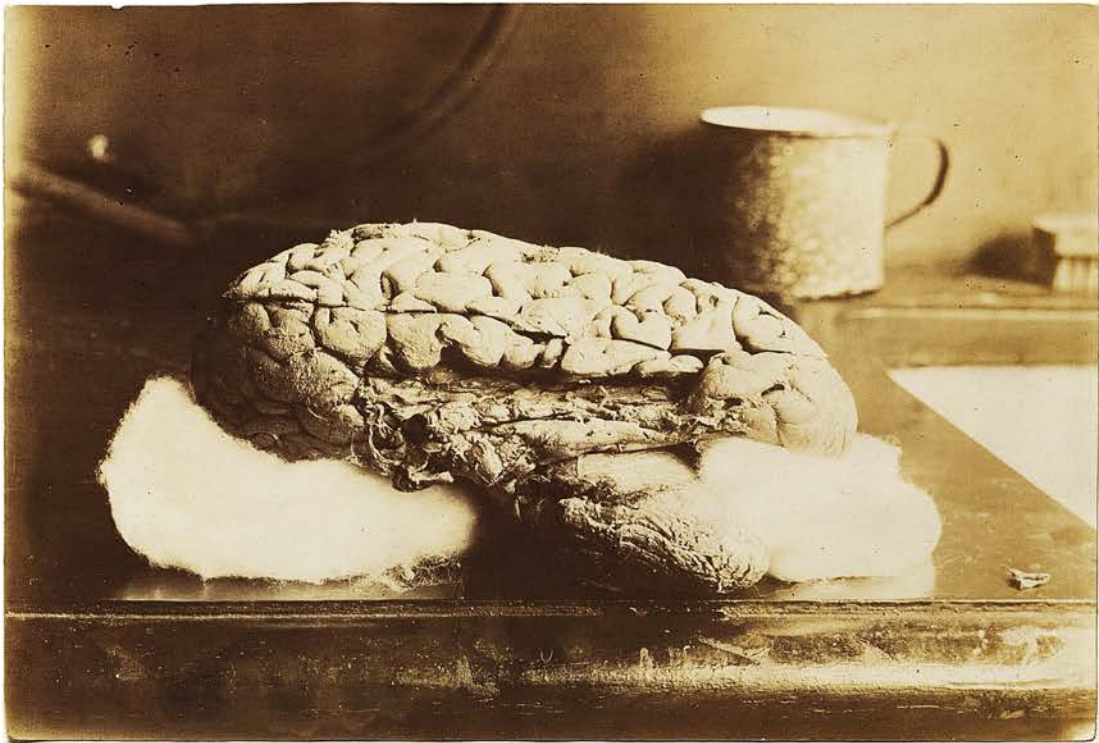
about 24 hours after the attack she was in her usual again although weak. From that date she remained in her usual health till the onset of her present attack, for which she was admitted into the Hospital in the semicomatose condition previously described.

Progress. Her temperature on admission was 101.4° but after an ice cap was applied to her head it gradually fell and on the morning of the next day it was normal, rising again in the evening to 99.8° falling next morning to normal, and rising again in the evening to 100.4° .

She gradually became weaker, and died comatose on the 29th September, four days after admission.

The following is the report of the Post Mortem examination of the head.

On removing the skull cap, the dura is seen to be thickened, and vessels in pia mater congested membranes are oedematous looking, and much fluid is found in arachnoid spaces between the convolutions. A large collection of clear fluid in the Sylvian fissure on the left side where it is seen that the upper two convolutions of the temporo sphenoidal lobe have almost entirely atrophied, their place being occupied by a large quantity of fluid, the surface vessels being much congested over this area. The atrophic process passed back to the angular



CASE. III.

Destruction of the first and second and the greater part of the third convolution of the Temporo-Sphenoidal Lobe, and a part of the angular gyrus and supra-marginal Convolution of the Left Hemisphere

Fig. XV.

gyrus and posterior part of the supra marginal convolution, but the part of these convolutions nearest the Sylvian fissure was also involved in this atrophy. The dura mater was adherent to cerebrum at various parts of the hemispheres especially over the vertex. The atrophy was quite evidently due either to an embolus or a thrombus blocking one or more branches of the Sylvian artery. The lower half of the third temporo-sphenoidal convolution escaped the atrophic process as also did the third left frontal and Rolandic areas, but the region of the motor areas although not visibly atrophic were not quite so full as the other side. Nothing further was seen on slicing the brain. A good photo of the left hemisphere is here inserted, which gives a good idea of the precise extent of the disease.

Fig. XV.

It was taken after the brain had lain some time in spirit and the third temporo-sphenoidal convolution had contracted relatively a little more than the rest of the specimen.

From the history of this case it will be seen that she had 17 years ago, what was apparently very marked hemiplegia involving the face, arm and leg on the right side, along with aphasia.

The attack was probably either thrombotic or embolic in character, more probably the former for these reasons:-

1st. There was no cardiac lesion which might have given rise to an embolus.

2nd. It came on after child-birth, as is not uncommonly seen, the usually accepted explanation being that although it is very improbable that an embolus of sufficient size to block a cerebral artery could pass from the venous circulation in the pelvis through the pulmonary circulation to the left side of the heart, and thence into the carotid artery, it is not quite so improbable that a very minute body might do so, and form the nucleus for the beginning of a thrombosis in a cerebral vessel.

She gradually recovered from the hemiplegia, the leg and arm recovering first, and ultimately completely so. On her second admission into Morning-side Asylum, six years ago, there are very careful notes taken of her motor condition, and it is seen from these that very slight motor impairment could then only be noticed in the face, the arm and leg having completely recovered. Three years ago she had fits, which apparently were more or less of the Jacksonian type, the motor movements being much more marked on the right side. The attack for which she was admitted into Leith Hospital was one of those congestive attacks which so commonly come on in old standing cerebral cases. There was marked congestion of all the meninges at the post mortem together

with considerable excess of cerebro spinal fluid. This attack she did not recover from, and she died a few days after admission. Summarising her symptoms and answering the twelve questions previously considered we ascertain that,

1. She could hear sounds.
2. She could not hear words.
3. She could not understand words spoken.
4. She could see objects, etc.
5. She could not see words, but could see figures.
6. She could not understand words written.
7. She could speak voluntarily, but used incorrect words.
8. She could not repeat words spoken.
9. She could not read aloud.
10. She could not write voluntarily.
11. She could not write to dictation.
12. She could copy.

1. 4. 7. and 12, are thus answered in the affirmative.

2. 3. 5. 6. 8. 9. 10. 11, in the negative.

Comparing these answers with the results we have already arrived at it will be seen that this case corresponds in the answers given to the last case we have considered, viz - a Pictorial Auditory and visual aphasia (Cortical Auditory and Visual of Wernicke) except that in this case the patient could copy whereas in the other one the patient could not copy. It is not noted in this case, however, how the patient was tested. In the previous case the patient could not copy written words. It has been found that some patients although they are not able to copy printed words into written words are still able to print words from copy, just as they would trace a map, a drawing, or a design. Whether this patient copied in that way or not the record taken at Morningside does not say. Later in this paper I shall describe a case of word blindness with agraphia who was able to copy in this way. That is he printed from printed copy and wrote from written copy. Amongst some of the most interesting of the facts of this case are the following:-

1. That although the patient lived for 17 years after the onset of her aphasia and although her hemiplegic symptoms disappeared her aphasia remained practically in statu quo. This is most interesting for various reasons. It was stated by Wernicke and the statement has been accepted by most

if not all writers on aphasia since then that Sensory aphasias often recover more rapidly than motor aphasias and the explanation of this was sought for in the fact that the right auditory word centre more readily took up the function of the left auditory word centre than the right motor centre took up the function of the left motor centre. It was stated that as speech was first acquired by the auditory centres the auditory word memories were not so exclusively confined to the left side as the motor word memories were and that the uneducated right auditory centre had more word memories stored in it than the right motor speech centre and more readily acquired a further expansion of its function when called on by a blotting out of the left auditory word memories. Although this may be the explanation in some cases, cases such as this, rather throw doubt on the theory because at the end of 17 years in this case the patient was much in the same condition as at the beginning although the post mortem revealed that the right side of the brain was intact. We have seen also that in the previous case I have recorded where the symptoms were almost entirely the same and where almost certainly the same regions were affected the patient made a rapid recovery.

On reading the literature of aphasia one cannot

but be struck with the number of rapid recoveries of all forms of aphasia. There are probably various explanations of such rapid recoveries such as that the aphasia may have been due to congestion or to anaemia of a cerebral area; to an embolus which had caught in one vessel and then got washed away into an unimportant vessel; to an embolus which owing to its shape was able to block a vessel at one time and on being changed in its position failed to block it entirely; to the circulation having recovered by means of anastomosing vessels; to the absorption of exudation or new growths of a syphilitic character such as endarteritis ^{or} Pachymeningitis; to the gradual absorption of small haemorrhages; to some toxic condition such as uraemia etc., impairing the function of cerebral areas; to a condition of dehydration; as in the case recorded by Chouppe (49) where a man out of work and travelling without getting water to satisfy his thirst fell asleep on the roadside. On being wakened up he was aphasic but Chouppe found in his pocket an Hospital ticket stating that the patient suffered from "polyurie simple". This gave the key to his condition and on the patient drinking freely of water he recovered his speech in a few minutes.

Are there functional sensory aphasias?

Many and various are the causes which have been

believed to cause aphasia and hysterical and functional aphasias have also been described. It has been generally recognised that there are many cases of functional or hysterical motor aphasia and hysterical and functional amnesia of nouns but true functional auditory aphasia has not I think received the attention it probably ought to have done. Wyllie (20 p.408) states. "Thus among the phenomena of functional aphasia may, as of occasional occurrence, be reckoned paraphasia, paragraphia, word blindness, and word deafness. But the occurrence of these phenomena is rare. The occurrence indeed, of word blindness, or of word deafness, ought generally to suggest to the mind of the physician the probability of an organic rather than a functional cause. In common cases of functional aphasia the leading and often the only phenomenon is amnesia verbalis (log-amnesia), the loss of the power of calling up words in the memory from within. Commonly the aphasia is purely amnesic, only in a few cases is it also agnostic."

These are the views of an observer of such experience as Wyllie but nevertheless one is struck on reading many of the cases of Sensory aphasia how well a functional cause would explain the rapid recoveries. Case II. is another of such cases, where

the patient without any motor symptoms and no other symptoms but word deafness and word blindness almost in one night recovered the functions of the auditory and visual word centres. The only objection to this theory is the fact that for two days there was slight rise of temperature but even that is not incompatible with hysteria or functional disturbance.

Wernicke's Case 1 (13) may fairly be considered to be a case of this sort. The following is a summary of the facts of this case. Susanne Adam was taken ill on 1st March. There was no loss of consciousness. There was complete auditory aphasia, paraphasia, occasionally correctly expressed herself, became intoxicated with a word, "begraben", was sent to an Asylum. Hearing was equal and good on both sides. Understood absolutely nothing from hearing it. Answered as much to a stranger's as to her own name, named objects rightly at one time but not at others. Sang a song without a book on hearing it sung by a patient. Had complete alexia. She progressed rapidly and on 20th April understood almost all that was repeated to her once or twice, speech was a little hesitating, read without stopping was not able to write to dictation but could copy pretty well the separate letters of the alphabet. Agraphia was then almost her only disturbance of speech.

I have selected this case of Wernicke's because it is a very well recorded case as well as being well known and was one of the cases that Wernicke drew his conclusions from as to the temporary nature of Sensory aphasias and the reasons of their temporary nature.

His conclusions on this point are stated at page 33 and are briefly:-

1. Motor aphasia cases show more general symptoms to begin with, which hinder diagnosis, but on the other hand the right side is not so ready to take on the function of the left side.

2. In sensory aphasia on the other hand, there is early compensation by the other side, and only slight general symptoms.

Now whatever the explanation of these rapid recoveries in sensory aphasia, I cannot believe that cases such as this one of Wernicke's (Susanne Adam) and my case (case II.) recover by the right hemisphere taking on the function of the left, as regards word hearing and word seeing. The cases were very like each other, but recovery in my case was more rapid and more immediate than in Wernicke's. Of course it is possible that the left auditory and visual areas, may have recovered from the organic lesion if it was organic, but such organic lesions

are not often so completely and suddenly recovered from as in my case. We know moreover that functional motor aphasias (Hysterical mutism) often recover quite as suddenly as they begin but in others are more gradual in their recovery. Later in this paper I shall describe an excellent example of functional motor aphasia which gradually recovered, as well as some others which recovered almost suddenly. Another argument against the right side taking on the functions of the left so rapidly in sensory aphasia, is furnished by this my second sensory aphasia case (case III) which remained in statu quo for 17 years, and the satisfactory fact in the case was that the organic nature of the disease, and the precise situation of the lesion, viz:- in the auditory and visual word centres, on the left side were verified by the post mortem examination.

I of course do not wish to deny that the right side may and does take up the function of the left through time and by a process of slow education. Such cases have been recorded and notably one by Wyllie, and the well known case of Barlow, ⁽⁵⁵⁾ where a boy with heart disease had aphasia with right sided hemiplegia, and after gradually recovering his speech, he had another attack which completely deprived him of speech as well as producing paralysis

on the left side. At the post mortem of this case two lesions were found, the first, the old lesion in Broca's convolution on the left side, and the more recent one in the corresponding part of the right hemisphere. This case is usually taken as conclusive evidence of the right hemisphere taking up the function of the left. But what I wish to state just now is that the process is probably almost always a slow one, and that those rapid sensory aphasia recoveries are probably due to some other causes, one of which causes is that some of the cases are originally functional and not organic in origin.

Passing however from this subject I shall take up a few of the other interesting points in this case (case III).

II. The case was twice confined in an Asylum.

It was stated by Wernicke that cases of sensory aphasia especially when there are no general symptoms are often considered to be insane, and are sent to Asylums. There is little doubt about the insanity in this case, but it is interesting to note from a Medico-Legal point of view, that the Medical Certificate stated the following facts, as being evidence of her insanity on the second occasion on which she was admitted. "She had obstinate taciturnity. Quite unable to give any account of herself. Does

not know her name. Talks incoherently". These symptoms, - without being too critical as to how a patient could have obstinate taciturnity, and talk incoherently at the same time - are quite what might be expected of a patient who was affected with sensory aphasia, but the patient need not necessarily have been insane.

III. The patient although she could speak voluntarily had a very small correct vocabulary. This is a point that has to be carefully investigated in all aphasia cases, because one is very apt to suppose that the cause of this difficulty in speaking correctly must be due to some impairment of Broca's convolution. More careful investigation however, reveals the fact that the correct vocabulary is small from impairment of the auditory word centre, so that the proper auditory word images cannot be recalled by the "ideational" centres. She could not often recall the correct word. She sometimes talked a great deal but the words were not quite correct, hence one medical certificate bore that "she talked unintelligible nonsense" and the other that "she talked incoherently". This is the symptom of paraphasia, which is such a marked symptom in conduction aphasias but is also always present in auditory aphasia proper. Case II. also showed this symptom

markedly and it will be seen that in the next case I have to record one of Conduction aphasia the symptom is very marked.

IV. The patient could see figures but not words. She could however, not calculate ("could not count up"). It is noted that this case on one occasion picked out a mistake in the figures in a baker's bill. When I consider cases of Visual aphasia I shall refer again to this difference between word and figure memories.

V. She possessed intact the language of signs, pantomime language. Her friends used signs in conversing with her and she used signs in conversing with them. She pointed to the height of her children to distinguish one from another. In referring to her brother, a plumber, she pointed to the gas bracket, etc. Sometimes this language of gesture or signs is lost as well as speech written and spoken, but often it is not, as in this case, (Case III) and also in Case II. The reception centre for such speech is probably in the occipital lobes, in or near the ordinary seeing centres, the production centre is in the centre for that part of the body used to produce the sign, the hand, head, etc.

VI. The very marked forgetfulness of names - amnesia of nouns.

She forgot almost all proper names even the

names of her husband and children, and could not recall the names of objects and things about her. This is a symptom I have said little about as yet, because I did not wish to complicate the schema which I have already drawn up. Both of these two auditory cases I have described had this symptom very markedly and it will be found on studying the literature of aphasia that there are very few complete sensory aphasias that do not show this symptom in a more marked or less marked degree. It has been recognised for many years that patients were more apt to have forgetfulness for nouns than for the other parts of speech. The usual explanation for this has been that although nouns of course are used almost in every sentence still the same noun does not recur often in ordinary conversation, whereas the other parts of speech recur again and again. Sentences are simply for the purpose of binding nouns in various ways to each other and the binding words are comparatively few in number and often recur. Many well known observers have adopted this view, but on the other hand, there are some who hold that there is a special centre for the storing of the names of objects, etc. Broadbent has written much on this subject and has published several most interesting examples of it (16. 50. 51)

From an examination of the anatomical arrangement of the connecting fibres in the cerebrum, he located the naming centre theoretically in the posterior part of the temporal lobe as he found that a large number of fibres converged to that region from the perceptive centres, the visual, auditory, tactile, etc., centres. Broadbent's papers, however, were written at a time when a word hearing and a word seeing centre had not been so generally recognised, and he does not so fully differentiate these centres from the naming centre. His cases besides being excellent examples of amnesia verbalis had also lesion of the word hearing and word seeing centres. The naming centre (or centres) is held by many authors to be the same as the "concept" or ideational centre (or centres). Bastian, Wyllie, etc., believe that in all probability there is no single centre for "concepts or ideas" but that many parts of the cortex are concerned in the elaboration of concepts and ideas. They also believe that there is no special naming centre apart from the ^{auditory word centre} ideational apparatus. Broadbent (hoc cit), Charcot (52), Kussmaul (15), and Mills (28), however, hold that there is a special naming centre but they apparently also believe that the naming and ideational centre is one and the same. That the ideational centre is

not one centre, but that probably many parts of the cerebral cortex act together so as to produce concepts or ideas and so act as one centre, seems the most feasible theory. The term "ideational centres" is therefore more correct than "Ideational centre." I think however that if speech be theoretically as well as clinically studied it will be seen that the probability is that there is a special naming centre apart from the ideational centres. The fact, however, that we have some cases where apparently all ~~th~~ the intellectual centres are intact, where the patient can think^{speaks} and act rationally, but on speaking has difficulty in recalling the names of objects, shows I think, that the ideational centres are distinct from the naming centre. It has been also shown for instance, that there are some cases where nouns could not be produced by the patient either voluntarily or by imitation, all other parts of speech being used freely. Such a case is recorded by Broadbent (51) "During the whole of his illness (5 years) he was scarcely ever known to utter a noun substantive, and if he did it was, so to speak, inadvertently and erroneously. Other words he said unhesitatingly and he would employ fairly long phrases, speaking them smoothly and naturally so long as a noun did not come in his way." Everyone^{knows}

how when the nervous system is tired and worn out either from fatigue or as the result of debilitating disease, the memory for nouns (proper names, etc.) is apt to fail even although the binding words (verbs, adjectives, etc.) in the sentence may be recalled quite easily. Of course the explanation of this according to some is as previously stated that the binding words are much more familiar having been used much oftener than nouns, but this theory does not satisfactorily explain such a case as Broadbent's where every noun substantive seemed to have been blotted out. A more satisfactory theory in my opinion would be one in which a special naming or noun substantive centre found a place. Such a centre must be very closely associated with the auditory word centre and the ideational centre or centres and on the receptive side must be connected with all the primary perceptive centres. The primary perceptive centres from which it could receive impressions would be the following. The centres for

Tactile Sensations.

Muscular sense.

Olfactory Sense.

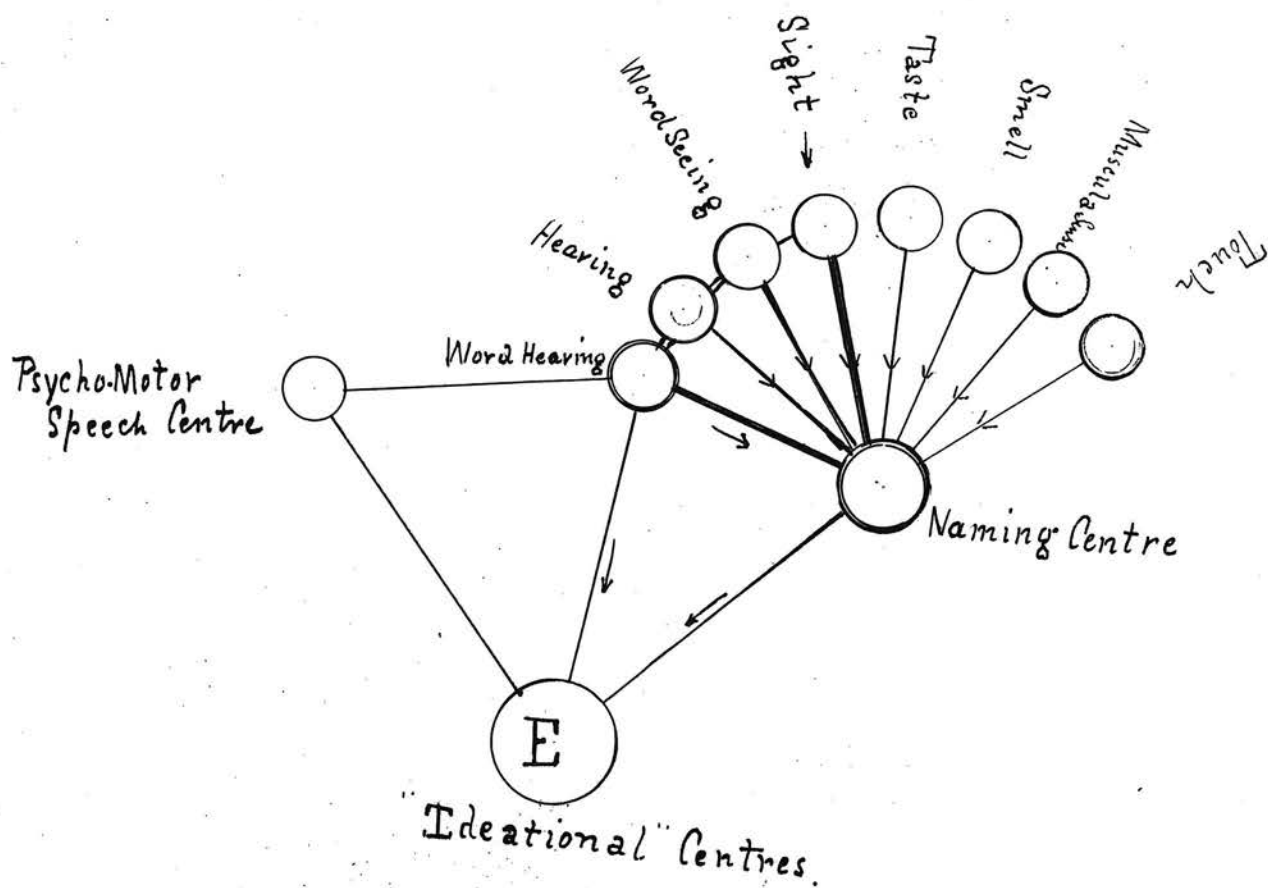
Taste Sense.

Seeing sense, including form colour, etc.

Word Seeing.

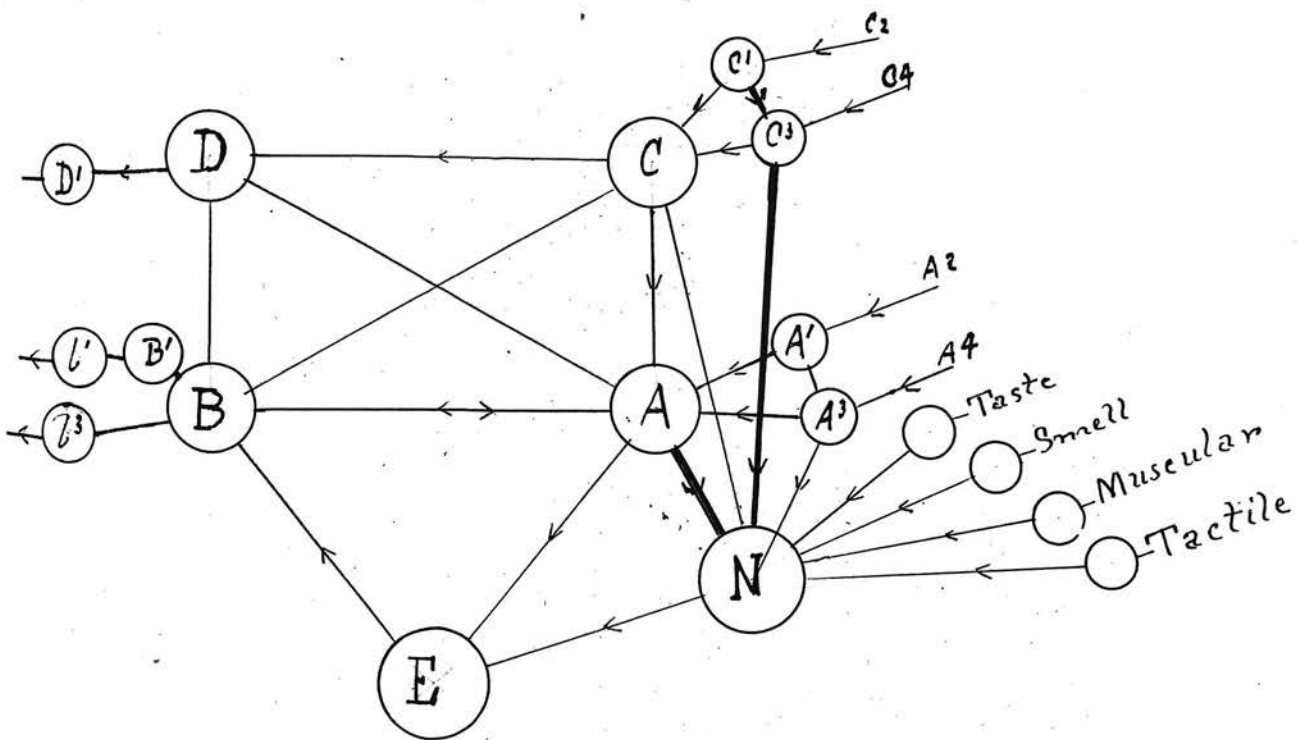
Hearing.

Word hearing.



Connections of the Naming Centre

Fig. XVI.



E = Ideational Centres
 N = Naming Centre
 B = Psychomotor Speech Centre
 D = Graphic Centre
 C = Visual Word Centre
 C' & C'' = Sight Centres
 A = Auditory Word Centre
 A' & A'' = Hearing Centres

Fig. XVII.

Fig. XVI is a diagrammatic representation of the connections of the naming centre.

In order to produce a complete schema of speech therefore, this diagram Fig. XVI would require to be added to the one I have previously drawn out, and the result is seen in Fig. XVII.

A. The Auditory word centre, a 1, a 3, The right and left auditory centres.

C. The visual word centre, c 1, c 3, the right and left visual centres.

B. The psychomotor speech centre.

D. The psychomotor graphic centre.

E. Ideational centres.

N. Naming centre, receiving impressions from A. a 1-a 3. C. c 1-c 3.

Taste, smell, muscular, and tactile ^{perceptive} centres.

Most objects receive their distinguishing name probably chiefly on account of the impressions of their form, colour, etc., which we receive through the visual perceptive centres. I have therefore drawn the line connecting the visual centres to the naming centre broader and heavier than the others, but for some objects most of the other perceptive centres send impressions to the naming centre, for others not so many. Take a violin for instance. The cerebrum receives impressions of a violin through

the nerves of sight (form, colour, etc.) through the nerve of hearing (recalling its sound, etc.) and through tactile and muscular sensations. All these impressions are received from the primary centres, (which it is to be noted are in both hemispheres), are associated in what I have called the naming centre with the auditory word image, or sound of the name of the object, received from the auditory word centre, in the left hemisphere only, and in educated persons probably also associated with the visual word image of the name, received from the visual word centre in the left hemisphere only, and the complete knowledge of the object, that is all we know of it from the sensory impressions including its name and the visual image of the name, can be transmitted to the ideational centre or centres or can be revived in the memory by the ideational centre or centres. The name and the other sensory impressions, however, can be recalled by one or more, of these sensory impressions as well as revived from within, for instance, if our eyes are closed and we hear a violin the sound at once recalls the name and the visual word image in educated persons; and at the same time the form, colour, etc., and other characteristics of it are recalled. Whether this is done by means of commissural fibres connecting

the various perceptive centres, or by a process of radiation from the naming centre, it is impossible to say, in all probability the various centres are linked in a most complete and complicated manner. For a full appreciation therefore, of the object, "violin" the naming centre must receive impressions from the visual, tactile, muscular, auditory, and word seeing centres, and these impressions are associated with the impression received from the word hearing centre, that is the word sound "violin" which we call the name of the object. On this theory the naming centre is a lower centre than the ideational centres but higher, although probably closely associated with the word hearing centre, and receiving impressions from probably all the other perceptive centres. For some noun substantives impressions may be received from one perceptive centre only, for others from two, for others from three and so on, but however few or many, perceptive centres, impressions may be received from ^{for} a particular noun substantive, these impressions are associated in the naming centre with the sound name of the object, the image of which sound name is received from the auditory word centre, probably in the immediate neighbourhood. From this naming centre these associated impressions can, as I said, be

transmitted to the ideational centres or revived from the ideational centres. How do noun substantives differ from other words, verbs, adjectives, etc. the binding words in a sentence? It will be seen that it is not necessary to receive any sensory impressions other than the ordinary word image, - and in educated persons the visual word image - of the binding words in the sentence in order to use these words in forming sentences. Such words are revived almost if not entirely, from within, that is in the ideational centres and probably the word is revived, if not in all in some persons at least by a direct route from the auditory word centre to the ideational centres or vice versa, without going through the naming centre or the centre in which the name is associated with the other sensory impressions of the object. From this argument it will be seen, that I have, to avoid confusion, omitted all mention of impressions received from the word production centres as they are common to all words; nouns, as well as binding words. As I have previously mentioned probably the most decided sensory impressions of most objects are received from the visual perceptive centres and therefore there must be many connecting fibres between the occipital lobes and the naming centre. Theoretically therefore, we would expect

that the naming centre would be in the posterior part of the temporo sphenoidal lobe, that is in a near neighbourhood to both the word hearing centre and the general visual perceptive centres in the occipital lobes.

On studying carefully many of the recorded cases of amnesia of nouns, where post mortems have taken place the lesions have usually involved either both the auditory word centre and the visual word centre or one of them so that it is very difficult getting sufficient evidence to show exactly where the naming centre is situated, if we now take for granted that there is such a special centre. Broadbent's cases proved quite distinctly that there was amnesia of nouns, if the greater part of the temporo sphenoidal lobe was destroyed, and they tended to show that in all probability lesion of the posterior part of that lobe produced the symptom.

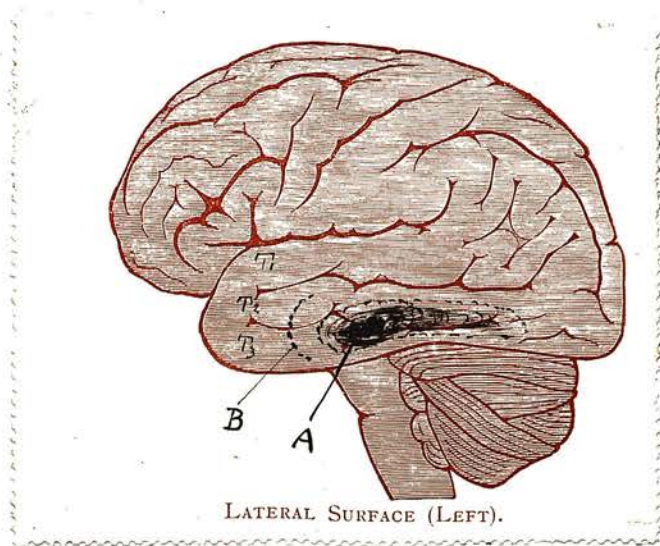
Recently, Mills (28) has recorded a case where at the post mortem there was a lesion limited to the third temporo sphenoidal convolution. The case goes a long way I think, to settle the precise position of the lesion which causes Amnesia of nouns. I give an abridged note of the case here. The patient - a woman, aged about 40 - had had cerebral symptoms for 5 years before her death, showing

gradual cerebral impairment, amongst the symptoms being a convulsion fit, word blindness, and verbal amnesia. She was first seen by Mills in consultation in July 1894. In April of that year she had had vertigo and from this time on "it had become almost impossible for her to name objects." In July there was no anaesthesia nor paralysis. She had an irregular left lateral homonymous hemianopsia. She was word blind, but not letter blind, she could name single letters slowly. She could not name objects, either from sight or touch. When a pencil, pen, scissors or purse were held before her or when she was allowed to touch them, she could not give their names, although she understood what they were. On one occasion she called the scissors, what I sew with, and the purse what I buy with. When such objects were named to her she would promptly indicate that the names were correct, and she could also as a rule repeat the names spoken in her hearing, but not always quickly, and occasionally she had considerable difficulty in repeating them. She used yes, and no, properly, and knew the use of objects, but could not give their names. She talked spontaneously but not freely, not using concrete nouns, or but rarely, and sometimes misplacing words. She became gradually more stupid, and died in September 1894.

At the post mortem a small nodulated, half

disintegrated mass about the size of a hickory nut, was pulled out of the brain substance at a position which corresponded to the posterior fourth of the 3rd temporal convolution. The surface of the 3rd temporal in its posterior half and to a much less extent of the 2nd temporal in the same region and of the 4th temporal presented a granular, slightly disintegrated appearance. On cutting into the temporal lobe, a tumour, hard and yellowish brown in colour was revealed. Its hardest and apparently oldest part was about the middle of the 3rd temporal and passing slightly into the 2nd temporal. The mass extended cephalad and caudad a short distance almost entirely in the white matter of the 3rd temporal gyre, but a soft nodulated more or less haemorrhagic condition reached caudad as far as the white matter of the middle of the occipital lobe, and cephalad to the junction of the first and middle thirds of the 2nd and 3rd temporal convolutions.

The parts chiefly destroyed were, the white matter of the 3rd, to a less extent of the 2nd, and to a still less extent of the 4th temporal convolution. Internally the roof of the posterior horn presented a slightly granular appearance. The disease almost certainly started in the 3rd temporal convolution at a point in a line with the posterior extremity of the horizontal branch of the Sylvian fissure. A diagrammatic representation of the area of the cortex involved is seen in the accompanying Figure. Fig. XVIII.



Naming Centre (after Mills)

Tumour of the third Temporal Convolution, indicating the position of the Naming Centre. A = Densest and probably oldest portion of the growth. B = Anterior limit of the lesion beneath the Cortex. Its limit on the surface are indicated by the dotted line

Fig. XVIII.

Here we have a very distinct localisation of the naming centre and the position is near to the place where we have theoretically supposed it would be, and where Broadbent long ago supposed it to be, from a minute study of the connecting fibres in the cerebrum. On examining carefully the records of many of the post mortems of cases of amnesia of nouns, such as those recorded by Wyllie and by Broadbent, I find that this region was more or less involved, in most if not all of them. In my case (case III) where amnesia of nouns was such a prominent symptom, this region was certainly also involved in the lesion, see photo. Fig. XV.

If therefore there is a naming centre separate from the auditory word centre as I hold there is from a theoretical, as well as a clinical study of speech, then all the clinical and pathological evidence points to that centre, being situated in the posterior half of the third, and perhaps also the second temporo sphenoidal convolution.

The post mortem examination in Case III. was also confirmatory of the position of the word hearing and the word seeing centres, as well as of the naming centres. If therefore we admit a naming centre, it will be seen that it is possible to have various lesions involving that centre or the fibres

connecting that centre with other centres, and these lesions might give rise to various symptoms according to the position of the lesion. This fact would account I think for the varieties we find of amnesia of nouns. We might have:-

1. An infra pictorial or subcortical naming centre lesion, (an infra pictorial or subcortical logamnesia), that is a lesion cutting off the naming centre from the incoming impressions, - chiefly as I have shown from the visual centres, - such a patient would not be able to name objects at sight, although if the centre itself was intact and not cut off from its connection with the ideational centres, and the auditory word centre, the patient might be able to use nouns voluntarily or repeat nouns heard.

The lesion is a subcortical lesion and Broadbent's first case (16) showed a lesion, viz:- in the white substance immediately external to the descending cornu of the lateral ventricle, just exactly where one would expect the course of the fibres from the visual centres to the naming centre to lie. A marked symptom in Broadbent's case was the inability to name objects at sight.

2. A pictorial or cortical naming centre lesion. (Cortical or pictorial logamnesia) which must probably be the most common. The patient would

not be able to name objects, or to recall names from within. Most verbal amnesia cases show this symptom.

3. A supra pictorial naming centre lesion
 (supra pictorial or transcortical ^{where the fibres} logamnesia) _^ between the ideational centres and naming centre are involved. Such a patient would be able to repeat names if the naming centre was intact, but would not be able to name objects at sight nor recall names in conversation. It is questionable however, whether this form exists apart from the previous form, type 2.

The whole subject however, of the naming centre as apart from the word hearing, and the ideational centres is still involved in doubt, but I believe that a true solution to the question is to be found in the lines of the theories, I have endeavoured to indicate.

CONDUCTION APHASIA.

Passing from Auditory Aphasias proper I shall now consider Conduction Aphasias, Leitungsaphasie of Wernicke comprising Type IV and XI viz:-

- I. Inter Pictorial Auditory-motor.
- II. Inter Pictorial visual-graphia.

Although theoretically as I have said, there are two types practically only one type is distinguished clinically. There are probably very few absolutely pure cases of conduction aphasia. Usually they present some disturbance either of the auditory or visual word centres. The following case is a good example although it shows also some word deafness.

Case IV. (Personal Observation.)

Conduction aphasia, some word deafness, marked paraphasia, and paragraphia, marked gibberish speech, marked word intoxication, marked amnesia of nouns, no motor aphasia, no involvement of the music hearing centre.

D. M. aged 60, consulted me on 19th July 1893 complaining of loss of memory for proper names and a peculiar flushed feeling coming on him occasionally. The first feeling of this sort had occurred about a

fortnight before at a Railway station. He lost his memory for a few seconds so that he could scarcely speak. He was going to the country at the time, and he remained in the country for a few days and had one or two attacks of the same kind each day.

This continued till I saw him. He had the last attack of this sort in my presence on the first occasion on which I saw him. During all this time he had more or less difficulty in recalling proper names, even the names of his nearest and most intimate friends. His memory for proper names was worst during and immediately after these attacks. The attack I saw him in was a short one but of the usual character. He was speaking, giving an account of his illness when he suddenly stopped for about half a minute and then said "that was one of my attacks." I noticed that he hesitated often in speaking for a few minutes afterwards especially at the nouns and more especially when he wanted to use some special noun or proper name. His friends stated that he had been failing in health for a year or so, and his present illness had really begun before he himself confessed to its having commenced. He was an exceedingly temperate and regular man in every respect.

Examination of his organs revealed nothing but slight impurity of the 1st sound of the heart. The only symptom apparent at that time in his nervous system was the slight difficulty he had in recalling proper names and nouns. He could read and write quite well. About a week afterwards he began to complain of a severe pain in his head and he showed a tendency to get worse in his memory. He was put to bed for a few days and not allowed to see any one. His condition now rapidly became worse and on the 2nd August the following was his condition.

He could read aloud comparatively well, making very few mistakes. He could not write a correct note voluntarily. He put in wrong words, and repeated the same word over and over again. He made also a great many mistakes when asked to write to dictation often putting down wrong words and repeating the same word. He could copy a paragraph perfectly. When asked to repeat such a phrase as "West Register Street" he said something that sounded like it such as "Westminster Street."

When shown a watch he said it was "a watch." When shown a pencil he said it was "a watch". When shown a watch-chain he said it was "a watch". When shown a button he said it was "a watch". In conversation he showed the same tendency to repeat the same word. He said for instance that he had

Volitional writing.

D Lu is committed
about fortnight a piece at least
about fortnight

D Lu about fortnight
throughout, I began two
months next week, and since then
I have again regularly

Fig XIX

over

Written to Dictation.

Receipts I began to engross receive
Receipts of various nature of yesterday ~~the~~ state

Copied from print.

Of all the manner of space, the the above history

difficulty in getting a full breath on going upstairs and then proceeded to talk about taking his dinner and instead of food or dinner he always used the word "breath." He sometimes did and sometimes did not know he had used the wrong word. He always knew he had difficulty in finding the word but after getting one he often seemed satisfied. Often he used the word that sounded like the one he wanted and often he used a sound like the word wanted, but not a word at all in reality. He had difficulty in counting (addition, subtraction, multiplication, etc.,) but this apparently was not from difficulty in knowing how much for instance 6 and 5 was as the difficulty in recalling the word eleven. He understood and used gesture language well. There was no disturbance of motion or sensation.

A specimen of his writing to dictation, voluntarily and to copy is seen in Fig. XIX. His voluntary writing was an attempt to write an account of his illness, paraphasia is the most marked symptom of it, as well as word intoxication. The words "about fortnight" were written twice in the first 3 lines and appeared again in the fourth line when he made another attempt to write. In the 5th and 6th line the word "next" appears three times. Some of his attempts at writing were not words at all, and the grammatical arrangement of the sentences as

they stand is very incorrect.

In his writing to dictation some of the words are also incorrectly written. The sentence that was dictated to him was,

"Dear Sir,

I beg to acknowledge receipt of your letter of yesterday's date."

The words "beg," "acknowledge" and "letter" are incorrectly written and his first attempt at "receipt" was also incorrect.

In copying he could both copy written writing and also could copy print into writing: a fact which shows that his word seeing centre was intact. The words seen reached his intellectual centres and he was then able to convert the printed letters into written letters. Later I shall describe a case where the patient could copy but only by tracing so that he always copied printed letters as they were printed and written letters as written. See Fig. XXI

D. M. gradually but very slowly got worse, his memory became more impaired, he used wrong words more frequently, so that in about three weeks he could scarcely make himself understood only being able to answer a question "yes" or "no" or short sentences such as "very much" "very bad", etc. He often gave practically the same answer to many

different questions becoming often intoxicated with a word and using it over and over again.

It was noticed also that there was a very slight dragging of the right foot as if he had not quite so much power in that leg. His right hand had also a little less power than the left and there was slight drawing of the face and angle of the mouth to the left.

August 25th. He was much the same as the last report. He could scarcely make himself understood, beginning sentences apparently all right but ending in a "mixture of words" the same word often appearing in many sentences.

1st September. Much the same but generally a little weaker and more staggy. The right side was slightly weaker, the right side of his face flatter. There was slight increase of knee reflexes on the right side. On being asked he gave his name all right. What do you do? A. Do I mean. I cant tell any more.

Are you 40? A. Fifteen,- fifty pounds fifteen shillings.

Were you out for a walk yesterday? A. No. I don't think so.

What had you for breakfast to-day? He muttered

a lot of words amongst which fifteen was recognised.

What had you for dinner? A. Crystal, good-a-look - good-a-look and other gibberish.

What is two times five? A. Three times to the right.

When asked to touch his nose he always opened his mouth but did touch it after some time.

When asked to touch the Doctor's ear he said "yes" and a lot of "gibberish."

Say "Grocer". A. "Yes."

Put out your tongue. A. "Yes."

Ophthalmoscopic examination of the eyes was very difficult on account of the difficulty in getting him to look steadily at anything. It was seen however, that there was optic neuritis of the right eye. The following particulars are supplied by one of his relatives.

"On two successive Mondays he has asked that the list of newly published books should be read to him from the "Scotsman" newspaper, the day of the week never having been told him nor that part of the paper near him. On Thursday, August 31st, on being asked if there was anything in the newspaper of that date he wished read to him he took hold of the paper and pointed to a biographical sketch of an old friend who had just died. He had always been fond of

music and during his illness especially so. For hours he will listen and never wish to move. Whenever he hears a wrong note played he winces. He was tested as to this; a wrong note being intentionally introduced and he detected it at once. Every Sunday he has asked that hymns should be played and on two different Sundays when "Lead kindly light" was begun he became emotional and broke down.

On September 11th I noted that he talked even more gibberish and that it was very difficult getting any intelligible answer to any question. He got gradually weaker from this date and duller intellectually and died on the 8th of October 1893. No post-mortem was obtained. Dr Wyllie, Edinburgh kindly saw the case on two occasions with me. From a careful study of this case it will be seen that the answers to the 12 questions I have previously drawn out are all in the affirmative although there were great imperfections in the performance of many of the acts to which the questions refer.

The only centres that were partly involved from the beginning were the word hearing and the naming or noun substantive centre. The disease in all probability began subcortically in the connecting fibres between the naming and auditory word centres

and the motor word centres, but nearer the former which were also partly involved at the beginning and ultimately almost entirely so. The most marked symptoms in the case were paraphasia and paragrammia which are such distinguishing features of Conduction aphasia cases.

"Word intoxication" and "gibberish" speech were also very prominent symptoms in the case, as well as marked amnesia of nouns. The visual word centre seemed to have escaped altogether but the connecting fibres from it to the graphic centre were probably involved. He knew the uses of objects but could not usually name them. Lastly, a very marked feature of the case was the retention of the power of hearing melody and of appreciating and understanding heard music.

One cannot, in the absence of a post mortem be definite as to the precise cause of the disease but the slow and gradual onset and progress rather pointed to some form of new growth, although the symptoms are not incompatible with acute softening or atrophy from a thrombus or an embolus. Towards the end of the life of the patient the motor areas in the neighbourhood of the fissure of Rolando on the left side became slightly affected probably from spreading forward of the disease. The position of the lesion

causing conduction aphasia is supposed to be usually in the floor of the Sylvian fissure and in the Island of Reil probably it primarily began a little further back in this case, viz., in the substance of the temporo sphenoidal lobe, subcortically to the Auditory word centre and naming centre hence the early symptoms of slight word deafness, and amnesia verbalis. In addition to being a conduction aphasia it was therefore an example of what I have called the 1st form of logamnesia, the symptoms of which I have previously indicated when I was considering the noun substantive centre. The distinguishing feature of this 1st form is the symptom of want of ability to name objects at sight from a subcortical lesion cutting off the naming centre from the perceptive centres.

VISUAL APHASIA, WORD BLINDNESS or CÉCITÉ VERBALE.

This form of aphasia has within the last few years been very carefully investigated and the two principal forms of it described by several well known French authors chief amongst whom are Dejerine and Sérieux. In a series of papers read before the Biological Society of Paris in 1891 - 92 (17 & 18) they brought forward cases which conclusively proved the separate existence not only of visual aphasia (Cécité Verbale) but also the existence of two distinct clinical and pathological forms of it. Little however had been done in this country on the subject until Wyllie (20) showed the position of our knowledge up to date. Recently (Dec. 1895) in an article contributed to the Lancet Hinshelwood gave an excellent resumé of the work that had been done on Visual memory and word blindness, and he contributed some clinical observations on the subject. From what I have already indicated in a former part of this paper it will be seen that there are two principal forms of word blindness (Cécité Verbale) and the distinguishing symptom between the two forms is the presence or absence of agraphia. Dejerine in his able contribution on the different varieties of

Cécité verbale divides the cases into two forms:-

1. Cécité verbale avec agraphie ou troubles très marqués de l'écriture.

11. Cécité verbale pure avec intégrité de l'écriture spontanée et sous dictée.

These two forms are two of those we have theoretically presumed to exist when we were considering the mechanism of speech.

1. Cortical Alexia or pictorial word blindness due to lesion of the word seeing centre (visual word centre) in the angular gyrus and supra marginal convolution. In addition to not being able to see words, patients with this form have also very marked disturbances of writing. They are not able to raise the visual image of the word in their memory as such image has been blotted out. This is Type IX lesion of C in schema.

2. Subcortical alexia or infra pictorial word blindness where the word seeing centre itself is intact but the centre is cut off from the visual perceptive centres in the posterior and internal part of the occipital lobes by a lesion involving the fibres passing from these centres to the word seeing centre. Such a patient although he cannot read can still write spontaneously and under dictation owing to the fact that he is still able to raise

the visual image of the word and letters in his memory. His ability to copy however is much disturbed. He copies letters written or printed just as he would copy a design, a map or any drawing. He copies ^{printing} printing as _^ and writing as writing. He does not copy printing into writing. This is Type VIII lesion of c1, C and c3, C in the schema. Fig XII. No case of the other theoretical form of visual aphasia Type X. Supra pictorial visual, (Transcortical Alexia of Wernicke) has yet been described and it is very questionable whether it exists apart from Type II. and III. Pictorial Auditory, and Supra Pictorial Auditory so that it may not further be considered.

Before proceeding to give clinical examples of these two types of visual aphasia it will be necessary to consider briefly the bearing of what we have already learned with regard to the visual receptive tract on the production of disturbances in speech due to lesion in that tract. We have already found 1st that each retina is bilaterally represented in the hemispheres. That the visual perceptive centres for the left half of the left retina and the left half of the right retina are situated in the left hemisphere. And the visual perceptive centres for the right half of the right retina and the right

half of the left retina are situated in the right hemisphere.

2. That the visual perceptive centres are situated in the occipital lobes in the cortical region on the internal surface of these lobes in the neighbourhood of the calcarine fissure. The centres for colour although not exactly in the same region are close to it. The lingual lobule and the cuneus may be taken as including the visual centres.

3. That the course of the optic tract from the anterior quadrigeminal body to these visual centres is in the radiations of Gratiolet.

4. That these visual perceptive centres are probably connected with each other by commissural fibres seeing that both halves of each retina require to act together. These fibres probably pass in the posterior part of the corpus callosum from one hemisphere to the other but they have not definitely been made out.

5. That there has been specialised a centre for the perception of written words which like the other speech centres is situated in the left hemisphere only, in the angular gyrus and posterior part of the supra marginal convolution.

It will be seen why it is necessary to have such a centre when one considers that the ordinary

visual centres in all probability besides enabling us to see the colour etc., enables us to perceive also the form, shape, etc., of objects, but this form or shape is simply as we would perceive a drawing, a chair, or the letters of a language unfamiliar to us such as Chinese. It is necessary for reading to have stored in the cerebral cortex the visual memories of special shapes and forms, - that is letters, and combinations of shapes and forms, - that is combinations of letters - or words - previously perceived. And in order that these visual memories may be associated with the auditory memories of the letters and words it will easily be seen that it is more convenient and economical for nature to store them in the "Zone of speech" in the left hemisphere. The visual memories of words are therefore situated in the angular gyrus and the posterior part of the inferior parietal lobule or supra marginal convolution. On this theory the most specialised part of this word seeing centre viz:- the memories of the longest and least common combinations of letters is situated probably furthest forward or at least not exactly in the same place as the less specialised part viz:- the visual memories of the more familiar words and single letters. If such be so it would explain the fact that some patients who may not be

able to read long and unfamiliar words are able to read letters and small familiar words, words that they apparently perceive as one object or form just as, they would a letter. It will be found that although a word blind patient is not able to read words he may be able to read figures. The explanation of this is as I have said probably because all the visual memories are stored not precisely in the same region, although another explanation may be that some of the more familiar and common visual memories may be imprinted also in the corresponding uneducated centre on the right side. Whether the visual perceptive centres alone - both angular gyri being destroyed - could enable an individual to see a letter a figure or small and familiar word such as "the" as a letter, a figure or "the" we do not at present possess sufficient knowledge to say absolutely definitely but in all probability it will be found that the visual word centre can be divided into various areas each having its own special memories stored in it. Such is the most feasible explanation of the clinical facts.

As I have said some patients although word blind can read figures. Both cases II. and III. showed this symptom quite distinctly and there are others in medical literature. Granted therefore that we have the two visual perceptive centres and

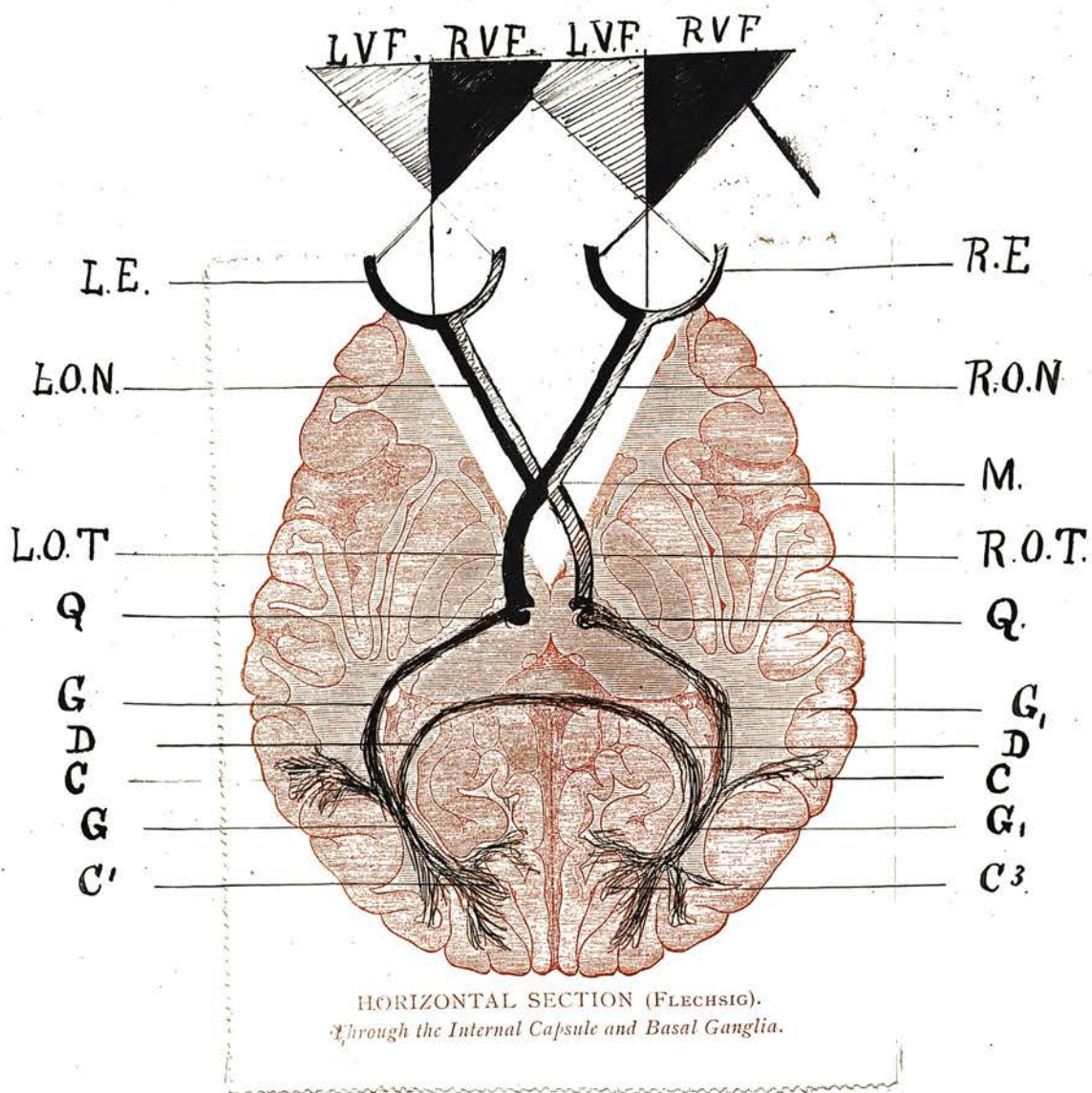


Fig. X X.

one specialised centre for letters figures and words both visual centres must be connected with the word seeing centre by fibres, the left visual centres directly, the right visual centres either directly or indirectly through the left visual centres. The accompanying diagram which is modified from the one in Dejerine's paper shows the course of these different fibres. Fig. XX.

C is the word seeing centre in the angular gyrus, cl is the left visual perceptive centre region in the occipital lobe and c3 the corresponding region on the right side.

D. the commissural fibres connecting these two centres.

G and G1 the radiations of gratiolet passing down from the occipital lobe.

Q the corpora quadrigemina.

L O T the left optic tract.

R O T the right optic tract semidecussating at M the optic commissure.

L E the left eye and R E. the right eye.

L.V.F. The left visual field,

R.V.F the right visual field.

From a study of this diagram it will be seen how lesion of the fibres between M the optic commissure and cl the visual perceptive centres on the left

side produces blindness on the left side of the retina of each eye, hemianopia and in the right field of vision right lateral homonymous hemianopsia and hemiachromatopsia or colour blindness and lesion in the corresponding fibres on the right side produces left lateral homonymous hemianopsia and hemiachromatopsia.

Lesion of the radiations of Gratiolet G thus produces those symptoms and lesion of the centres cl or c3 also the same symptoms.

Lesion of the fibres passing between the visual perceptive centres cl, c3 to C would not produce hemianopsia if they only were involved in a lesion but the radiations of Gratiolet would also be very apt to be involved so that right lateral homonymous hemianopsia and hemiachromatopsia is a very common lesion along with infra pictorial or subcortical word blindness as well as with Pictorial or Cortical word blindness if the lesion extends deep enough into the white substance of the occipital lobe.

Hinshelwood (53) has collected eleven such cases which with two others recorded by himself make 13 cases, as having all had this symptom along with word blindness. As however cases can occur without this symptom, it proves that the word seeing centre is separate from the ordinary visual centres. Of

the four cases of word blindness of my own observation 3 had not hemianopsia. Two of these latter had auditory aphasia as well as Visual aphasia and have already been described in an earlier part of this paper (Cases II. and III.) the other was a case of Cortical visual aphasia which I will describe a little later. The fourth case was not tested as to her visual fields.

I shall now give clinical examples of these two forms of Visual aphasia taking them in the order we theoretically considered them.

1st. Infra pictorial Visual aphasia (Wyllie).

Subcortical alexia (Wernicke).

Cécité Verbale pure. (Dejerine).

Case V. (Personal Observation).

Infra pictorial aphasia wordblindness, but not blindness to letters nor figures. Ability to write spontaneously but inability to read what she had written, very slight and temporary disturbance of the object seeing faculty (mind blindness), rapid and complete recovery.

M.R. aged 67 was engaged one day in March 1892 writing a letter to a friend when she suddenly discovered that she could not see the words distinctly, and that after she wrote a sentence she could not see what she had written. She thought that she was

losing her eyesight and consulted me about it. Her only other complaint was that she had difficulty in recalling proper names and nouns, as well as slight impairment of her memory for events. The friend to whom she had written, on receiving her letter, wrote back and asked her what was wrong with her as she had repeated some sentences and words in the letter over again. Shortly before writing this letter, on her servant, who had been with her for some time, coming into the room, she turned to her niece and asked "what strange woman that was?" In a few minutes however she was able to recognise the servant and every other object about her and never had a return of this.

On testing her eyesight I found that she could see objects quite well, that she saw letters well and could name them slowly but that she could not put them into words. When however I spelled short words aloud for her she could pronounce the word from the sound but often in a slow and disjointed manner. She herself could read in this way, viz:- by reading the letters aloud and pronouncing the word from the sound of the letters but this could only be done with the smaller words. She could write voluntarily but she could not read what she had written, although she could make out the letters

and figures. She was not tested as to copying or writing to dictation but probably the latter was quite retained as she had no word deafness. She could hear quite well and could converse quite well, so that there was no impairment of her auditory and motor word centres. These symptoms persisted for two or three days and then they gradually passed off so that in three weeks she had quite recovered except that her memory was slightly impaired, for a little longer period. She is now four years afterwards, in good health and has had no return of her complaint.

Answering the twelve questions I have previously drawn up.

1. She could hear sounds.
2. She could hear words spoken.
3. She could understand words spoken.
4. She could see objects, etc.
5. She could not see words written or printed.
6. She could not understand words written or printed.
7. She could speak voluntarily.
8. She could repeat words spoken.
9. She could not read aloud.
10. She could write voluntarily.
11. Probably she could write to dictation (but not tested).
12. She could probably copy as from a design.

It will be seen that the answers correspond to Type VIII, and it was therefore a very typical case of cécité verbale pure, or infra-pictorial visual aphasia.

It is interesting to note that she had her powers of writing so well retained that she had actually been writing a letter when she noticed she could not read what she had written. There is little doubt about the fact that her trouble had come on before she started to write this letter, as she had shown a very slight form of *cécité psychique* or mindblindness before it. This is a form of disturbance allied to aphasia which I will have a little to say about later.

Another interesting fact in the case is that the patient had retained the power of reading letters and was able to read short words, by pronouncing each letter she heard the sounds, and repeated them combined into a word. I have not seen this power taken note of in any of the recorded cases.

The patient made a very rapid and complete recovery and in this respect her case is very like Case II, an auditory and visual aphasia case, and the remarks I made about functional and temporary aphasias in connection with that case, are equally applicable to this. Alongside of this case, of my

own observation let me now place a note of the case of Cécité verbale pure recorded by Dejerine, in a paper (17) which I have referred to on several occasions previously. It was the case that definitely settled the question as to whether, there were separate pathological lesions corresponding to the two clinical forms of Cécité verbale. The paper is a masterful exposition of the whole subject as well as a model of a clinical observation.

Dejerine's Case. The patient a man aged 68 was under observation for four years, first by Landolt who recorded it in the work published in Utrecht in 1888, on the occasion of the Jubilee of Donders and dedicated to him. The case was sent to Dejerine in 1887 and between then and 1892, he was seen frequently. The patient was a highly cultivated and intelligent man, and during these four years he was incapable of reading manuscript or printing. He wrote papers without being able to read what he had written. The word blindness was totale (cécité litterale et verbale), and was accompanied by right lateral homonymous hemianopsia and hemiachromatopsia. He also had complete blindness for the notes of music, although he had had a good knowledge of music previously. But although he could not decipher a single note of music, he sang himself and listened

frequently to his wife playing or singing opera music to him. Spontaneous writing and writing to dictation were perfect and easily performed, although the letters were a little bigger than before; just as any of us would write, if our eyes were closed.

On the contrary the act of copying was defective, he could only copy with each letter distinctly before his eyes and traced simply as a design. The letters were badly formed and it was difficult believing that the same man wrote the specimens from copy, as did those to dictation and spontaneously. He copied printed differently from written letters, in other words he printed in copying printing.

He had preserved intact the power of reading figures and was able to calculate both mentally and on paper. He never had the least symptom of word deafness (*surdité verbale*), and never except during the 10 days preceding his death, did he show the least trouble in speaking. His interior language was normal. He thought in spoken speech and heard mentally the words sounding in his ear, when he wished to write spontaneously, he heard the word and then saw it mentally. The visual image of the letters were intact in his interior language, the integrity of writing spontaneously and to dictation showed this. When one took his right or his left

hand or his foot and traced passively in the air the form of letters he recognised and named them. He was able actually to read letters by tracing them with his fingers in this fashion, by means of his muscular sense. In January, 1892, he was seized suddenly with very pronounced paraphasia and complete agraphia. He was incapable of writing the smallest word or a letter and could only scratch lines without distinct form. He died 10 days afterwards having preserved up to the end all his intelligence and the perfect knowledge of spoken speech having not the least word deafness (*surdité verbale*). From the clinical history Dejerine draws the following conclusions. The clinical history is made up of two stages.

During the first stage which lasted four years the patient presented the clinical picture the purest that one could imagine of the second variety of *cécité verbale* viz:- *Cécité verbale* pure without any alteration of spontaneous writing or writing to dictation.

During the second stage which lasted about a dozen days he had complete agraphia with paraphasia along with the *Cécité verbale*. In this second stage the clinical picture coincided with that of the first variety of *Cécité verbale*. *Cécité verbale* with

marked alteration of writing. (It is to be noted that Dejerine's first variety is my second variety and vice versa.)

At the post mortem pathological changes were found which accounted for these two different stages.

The first an old lesion occupied the occipital lobe and more particularly the convolutions at the extremity of the occipital lobe, the base of the cuneus and also the lingual and the fusiform lobules. The convolutions of this region were small and atrophied. The lesion was continued into the white substance subjacent and penetrated in the form of a wedge reaching the occipital horn of the ventricle. The optic radiations were degenerated and atrophied and the patch had destroyed the grey substance of the convolutions which lie alongside of the posterior part of the internal temporo occipital fissure.

This lesion was therefore situated in the visual cortical areas. The other lesion of recent date occupied the angular gyrus and inferior parietal lobule, and thus accounted for the wordblindness with agraphia which came on during the last few days of the patient's life.

This case of Dejerine's besides giving an excellent clinical picture of both of the forms of visual aphasia is quite conclusive as to the position

of a lesion producing each of the forms.

From this case it will be seen that the word Subcortical as used by Wernicke is a confusing term and that the word infra pictorial as suggested by Wyllie is a much more suitable one, because although the patient had subcortical wordblindness the post mortem revealed a very extensive cortical lesion in the visual perceptive centres. No doubt the effect of this lesion was the same as if the lesion had alone been limited to the white matter underlying the cortex at the angular gyrus. All that is necessary to produce Wernicke's subcortical wordblindness or Wyllie's infra pictorial visual aphasia or Dejerine's Cécité verbale pure is that the angular gyrus be cut off from receiving impressions from the perceptive visual centres on the same and the opposite side. It matters not whether that lesion is in the cortex so long as the effect is the cutting off of the angular gyrus from receiving incoming impressions from the nerves of sight. The same objections however do not apply to the term Cortical because in Cortical wordblindness the cortex at the angular gyrus must be involved in the lesion.

The following case is an example of the second type of visual aphasia, the type which Dejerine's case had during the last 10 days of his life.

Cortical Alexia (Wernicke).

Pictorial Visual Aphasia (Wyllie).

Cécité verbale avec agraphie (Déjerine).

Type IX. (lesion in C. Fig. XII).

CASE VI.

(Personal observation).

Word blindness with agraphia voluntary and to dictation; able to copy, but only by tracing the letters; no word deafness; no motor aphasia; considerable motor enfeeblement; and increase of reflexes.

The following are the notes of the case taken by Dr Hill Buchan, House Physician.

R.R., aged 24, a worker in a biscuit factory, was admitted to Leith Hospital on March 31st 1896, complaining of weakness, difficulty in walking, and general shakiness.

His parents are alive and healthy. Two brothers are alive and apparently healthy in body and mind. Eight of his brothers and sisters died when quite young. One from consumption, when quite a lad, who had taken fits up to the age of 14, and could never speak properly.

Personal History. The patient's mother says that he could never speak very distinctly. He

knew what he was going to say but his pronunciation was defective. Till he reached the age of 5 he was always delicate. A doctor is reported as having said that he had consumption of the bowels. He never had fits, never had scarlet fever nor rheumatism. He was five years old before he began to walk. He went to school at the age of eight. He was quite bright and sharp mentally and used to play about with other children and could do everything like them but was a bad speaker. According to the statement of his teacher, he could read but looked always a little "queer." When 14 he went to work in a biscuit factory, and was five years at this occupation, after which he became a painter in a shipbuilding yard. He was two years there when he took a fit. The day before he had got accidentally shut into a confined place, exposed to vapours of turpentine and naphtha and when he was got out he was very dazed and sick. He was better next day, but on coming home from his work he took a fit. He fell down and became unconscious, and kept moving his arms and hands about in a "waving" manner. He did not bite his tongue. This moving his hands and arms continued for a day as also did the unconsciousness. He did not recognise his mother and did not take any food. For three days he could not move

his legs at all. After this the power gradually came back, but he was unable to walk for a fortnight, and then only with difficulty. He remained dazed and stupid for some time. About three months ago after a wetting, he began to be troubled with pains shooting up from the ankles to the knees. These attacks since then have come on occasionally, and when they do come, he says the legs become quite stiff for five minutes, after which the pain passes away.

State on admission. He is a lad of rather poor muscular developement. His general expression of face is rather stupid looking. His palate is fairly high arched.

Nervous system. Sensory. There is very slight tenderness on precussion in upper part of the spine. The patient does not at present complain of pain, but says that he occasionally is seized with pain shooting up from ankles to knees. There is no paraesthesia. General tactile sensibility and temperature sense are unimpaired. His muscular sense is not only not lost but it is probably exaggerated. When he is asked to touch his nose, he does so correctly, but the movement is rather sudden.

Special senses. His hearing is quite acute, taste good. Vision for objects, etc., is good,

but he is word blind. Nothing abnormal revealed on ophthalmoscopic examination, no nystagmus.

Reflexes. Plantar, cremasteric, and abdominal, all exaggerated. On tapping forehead marked reflex of eyelid obtained. Pupils react to light and accommodation. Deep reflexes are also much exaggerated, both ankle and knee clonus being easily obtained. No disturbance of organic reflexes.

Motor. No paralysis but weakness of muscles. The grasp of both hands diminished, but the right more than the left. Dynamometer gives 25 K. with left, and 15 with the right hand. Muscular power in right leg less than the left. On making him twitch his face, the muscular action much more marked at the right angle of the mouth than the left, which is scarcely twitched at all. No marked difference on asking him to show his teeth. He puts out his tongue straight and it is slightly tremulous. His walking is very defective. He plants his heels down almost like an ataxic person, but all the time the limbs are in a sort of spastic condition and clonic spasm readily appears if he stands on front part of his foot. He can turn round fairly quickly. There is no nystagmus.

Speech. Motor. (a) Patient speaks in an indistinct slurring manner, no "staccato." There

1. W M R

Dictation of words "To & Man."

Russell R

Voluntary writing of name "Robert Russell."

O

Dictation of letter "O."

1 2 3 4 5

Dictation of Figures "1, 2, 3, 4, 5."

1 2 3 4 5

Voluntary writing "1 to 5."

NORTH

Copy of printed word "NORTH"

North
Ratt

Copy of the word North as written above.

Fig XXI.

is a trace of fine trembling movements at the angles of his mouth at times. Test sentences are badly pronounced.

Graphic. A Specimen of his writing, voluntarily, to dictation, and to copy, is seen in Fig. XXI.

It will be seen that there is almost complete agraphia in his voluntary writing and writing to dictation. The only letter that can be detected in his name is R. He can write some figures although very imperfectly. In copying he simply traces each line. He copies writing as writing, and printing as printing, putting in every peculiarity of the letter he is copying from. He was tested by writing frequently, and always the same result was obtained. He was always more able to copy plain printed letters than letters as written. This is a point I will refer to when I consider Agràphia.

Sensory. (a) He can hear and understand spoken words perfectly, there not being the slightest trace of auditory aphasia.

(b). Vision for words. He is quite unable to read words. He can sometimes make out letters, and very occasionally a small word. He can read figures more easily.

He was tested with the following:-

1 2 3 8 12 10, he said 1 2 3 8 12 10.

100 he said 100.

1500 he said 105

10000000 he said 1000.

He said he understood what he read, although he could not read aloud, but on practically testing him it was found that this was not so. For instance the following sentence was printed and shown to him; -
"PUT OUT YOUR TONGUE." He said it was his own name.

Intelligence. Below normal. His memory seems fairly good. He is quite sensible in his speech, but he seldom begins conversation. He has no delusions. Is shy in manner and somewhat sensitive as to being examined, neither depression, nor exhaltation, nor ideas of grandeur.

His other systems seem all normal. He was treated by rest in bed and given Iodide of Potash internally in 10 grain doses. He showed signs of improving in his powers of reading and writing, and at this date 24th April 1896, the improvement is well marked. He is much brighter looking, can^{now} read small words, and knows all the letters. He can also write a few letters, but the improvement in his powers of reading is greater than in those of writing. He is still in Hospital, under observation.

Remarks. This case is one of very considerable difficulty as to diagnosis, and at the present stage of the disease it is very difficult to be definite as to the precise nature of the lesion causing the symptoms. He presents many of the symptoms of multiple cerebro-spinal sclerosis, but there are two symptoms commonly present, which are absent in this case, viz: nystagmus and "staccato" speech. What also is rather against it being a case of multiple sclerosis is the symptom of most importance to us here, namely word-blindness, and the fact that that word-blindness is due to a cortical lesion. The Cortex is not very frequently involved in the early stages of multiple sclerosis, the sclerotic patches being more limited to the white substance, and involving the conduction fibres rather than the nerve cells. We are enabled therefore, to say in this case from our study of aphasia that the lesion producing some of the symptoms is situated in the cortex, and as most, if not all of the other symptoms could be produced by involvement of some of the other and notably the motor areas in the cerebral cortex, probably the same kind of lesion produced all the symptoms. This lesion I believe to be of the nature of pachymeningitis of a Chronic nature, with probably adherence of the meninges to the cortex.

The patient also shows some of the symptoms of general paralysis, but the distinct involvement of a limited cortical area is rather against this view also.

Into the whole question of the diagnosis in this case, I have no intention of entering, as all we have to deal with here are the speech symptoms, and these symptoms, as I have said, helped very much in the accurate diagnosis of the case.

He was an imperfect speaker, but he had always been so, although probably he had not quite so complete control over the movements of his articulating apparatus as he used to, owing to the excessive irritability of his motor apparatus from increased reflexes, due probably to motor enfeeblement of his cerebral motor areas, and perhaps also from patches of descending sclerosis as a result. Still there was nothing in the least of the nature of motor aphasia. He had no auditory aphasia. But had distinct, almost complete, word blindness, *cécité littéraire et verbale* with *agraphia*.

Answering our twelve questions.

1. He could hear sounds.
2. He could hear words spoken.
3. He could understand words spoken.
4. He could see objects.

5. He could not see words written or printed.
6. He could not understand words ditto.
7. He could speak voluntarily.
8. He could repeat words.
9. He could not read aloud.
10. He could not write voluntarily.
11. He could not write to dictation.
12. He could copy very imperfectly, and only by tracing lines.

This as will be seen exactly corresponds to Type IX. Cortical or Pictorial Visual Aphasia, the patient being exactly in the same condition as Dejerine's case was during the last 10 days of his life, except that there is no trace of paraphasia in this case, the cortical area being apparently very neatly picked out by the lesion. It is very rarely that a lesion is found so limited, as in a large number of cases the auditory word-centre is also involved and word-deafness therefore, often accompanies word-blindness. Cases II. and III. are two examples of this, and they are almost as typical cases of cortical word-blindness as they are of cortical word-deafness. In case III as we saw from the description and the photo, the post mortem revealed that the lesion had just reached to and involved the angular gyrus and aupramarginal convolution so

that that case supports the theory that these areas are the seat of the word-seeing centre. Before leaving the subject of Visual aphasia it is necessary to refer to a few cases that have shown symptoms which have been described under the name of:-

Object Blindness, Mind Blindness, or Cécité Psychique.

Not many cases have yet been described, and we do not know as yet a great deal about it. Charcot (52) described one very interesting case. Sérieux (18) another in which he had a post mortem examination. Gogal a third, also followed by a post mortem examination, and Hinshelwood (53) a fourth.

Personally I have not seen a typical case of this, but Case V. apparently had cécité psychique slightly for a short time as "on seeing her servant come into the room, she asked what strange woman that was." In object or mind blindness the patient is not able to recognise objects, persons, etc., which used to be familiar to him. The room seems strange, the streets, roads which he ought to know are quite strange. He does not know the uses of objects, etc. In Gogal's case (quoted by Wernicke) the patient bit into the soap, micturated into the wash hand basin, looked on jugs, thermometers, etc., as strange objects. In an earlier stage he could

neither speak nor understand what was said. At the post mortem there was found a lesion in the posterior part of the first temporal convolution, and behind the fissure of Sylvius and a similar condition in the third left frontal, the operculum shrivelled up, and the Island of Reil exposed. On the outer part of the surface of the occipital lobe on the right side and exactly on the tip, there was a yellow patch of indrawn cicatrix with superficial softening. In Sérieux' case there was word-blindness and agraphia, and also word-deafness and paraphasia, as well as object-blindness. She could not recognise her relatives and friends, their faces seemed changed just as if they wore masks, objects also she did not recognise, although not to so marked a degree as faces. At the post mortem, on the left side there was a patch of softening in the supra marginal convolution, and a limited patch in the first temporal; on the right side there was a patch of softening involving the supra marginal convolution and angular gyrus, and posterior part of first and second temporal convolutions. These are the only two post mortems of cases of *cécité psychique* which I have been able to find in Medical

literature, and in each of these cases it will be seen that there was a bilateral lesion, viz: - a lesion both in the right and in the left hemispheres, and it is therefore supposed that although the visual memories for words, etc., are stored in the left hemisphere, that the less specialised visual memories are stored in both hemispheres. In order therefore to produce mind blindness it is necessary to have a lesion in both occipital lobes, probably in the neighbourhood of the angular gyrus or the cortex more posteriorly, hence the rarity of this form of blindness, and the few cases of it that have been recorded.

4. APHEMIA. MOTOR APHASIA. BROCA'S APHASIA.

We now pass from lesion of the receptive speech centres and conducting fibres to lesions of the production speech centres. Motor aphasia includes three of the Types I have previously sketched, Types V, VI, and VII.

They are 1. Type V. Supra pictorial motor aphasia (Wyllie).

Transcortical motor aphasia (Wernicke). Lesion of E.B. in schema.

2. " VI. Pictorial motor aphasia (Wyllie).

Cortical motor aphasia (Wernicke).

Lesion of B in schema.

3. " VII. Infra pictorial motor aphasia (Wyllie).

Subcortical motor aphasia (Wernicke).

Lesion of B, b1, & B, b3 in schema.

Of the supra pictorial form there are one or two cases recorded in medical literature, notably one by Lichtheim (44) and one by Hammond (48). As will be seen, by consulting the answers to the 12 questions, under this form it differs from the usual Broca's type of motor aphasia in that the patient can

repeat words, can read aloud and can write to dictation. The only symptoms present are the loss of power to speak voluntarily and to write voluntarily, which are symptoms of all motor aphasias. I have not had any cases of this type under my care.

The second form Type VI. pictorial motor aphasia is probably the best known of all the aphasia types. It was the form described by Broca and his cases were excellent examples of it. The lesion producing it has been proved by many cases to be situated in the posterior part of the third left frontal convolution, the psycho motor speech area of Broca. The following three cases are very good examples of this type. The first was an organic lesion the second a most interesting case of a very temporary nature due to functional disorder of the psycho motor speech centre, in a case where albumin was present in the urine. The third also a very temporary form, probably due to a functional cause.

Case VII. (Personal Observation). Motor aphasia, Hemiplegia, gibberish speech for four years before death.

M.P. aged 91 was seen by me on 12th Nov., 1895, four years after having had a shock of paralysis. During that attack she was also seen by me and the following was her condition:-

She had complete paralysis of the right arm, very marked right facial paralysis, involving most decidedly the angle of the mouth, the lips and the tongue. There was also distinct paralysis of the right leg. She had also complete motor aphasia. She could not speak more than one or two familiar or common words, although she gave utterance to one or two sounds which were not words. She saw and heard quite well and understood what was being said to her. Four years afterwards, 1895, the following was her condition. The movements of the right leg were slightly impaired, there was also distinct impairment of the movements of the right arm and hand, and there was also distinct contracture of the same. Right facial movements were very slightly impaired. She could put out her tongue and it did not point distinctly to either side. During these four years she had been able to say very few words, occasionally "yesey", "aye", "no." She articulates quite well but the word she articulates has usually no meaning, as for instance "bitty, bitty, bitty," when she evidently wanted to say a few words. When asked a question she answered at once, but the sounds used were not intelligible words. Her hearing was very acute, and she saw and understood everything that was going on. On account of the paralysis of her arm it was

impossible to ascertain whether she could write or not. She died shortly afterwards from bronchitis. No post mortem was obtained.

This case was one of a common form. It was the usual right sided hemiplegia with aphasia. She had also a slight degree of paraphasia and "gibberish" speech. It is interesting to note^{that} during four years although there was considerable improvement of the motor power on the right side that practically the aphasia symptoms remained the same. The right psycho-motor speech centre had not taken up any of the functions of the left side, there had been no education of the "uneducated" centre of the opposite side. Probably the age of the patient (87) had a determining influence on this, but as I have indicated in a previous part of this paper it has generally been believed that motor aphasias do not so readily recover, by the education of the right uneducated centre as sensory aphasias do. In marked contrast to this case in respect to the duration of the aphasic symptoms were the two following cases.

Case VIII. (Personal Observation). Pictorial motor aphasia temporary in duration and recurring on two occasions in a patient with albuminurea.
Complete loss of the power of producing words and even sounds of any kind except a groan, complete

agraphia, no auditory aphasia, no visual aphasia.

H.E. aged 28 whilst recovering from a dysenteric attack as result of malaria contracted in South America, was seized with aphasia. He had had dysentery for several weeks and was gradually getting better being able to be up and out, when one day, 3rd April 1895, he partook very freely of food, a large quantity of which was of a nitrogenous kind. He drank a large quantity of strong tea, and then went to bed. In the morning about 7 o'clock on his friends going to his room, they found him lying in bed in a rather stupid-looking condition, with his jawsclenched. He understood what they said to him but he did not speak. He pushed his finger into his mouth, and tried to make himself sick as he appeared to think that would relieve him. I saw him about 9.30 a.m. and found him in bed, slightly stupid-looking with jaws clenched. When asked he moved his arms and legs about as requested, and pressed my hand with his, when I tested his grasping power, which I found to be normal. His arms showed a tendency to remain in the position in which they were put -(catalepsy). On asking him to put out his tongue, he indicated that he could not open his mouth. On attempting to put the handle of a spoon between his teeth, I found I could not force open

his jaws. Occasionally he gave expression to a very slight groan, but never made the slightest attempt at a word. He heard and understood words quite well and indicated that he could understand writing and printing, when given a pen and paper. He held the pen as if he was going to write, but never used it in any way even to draw a line. Mustard was applied to the back of the neck, and over the epigastric region where he complained of some pain. About 1. p.m. of the same day he took what was apparently a mild form of fit. He moved his arms and hands about, but did not clench the hands except once or twice quite spasmodically. For the greater part of this fit the hands were quite open. There was distinct twitching of the face more to the right than to the left side. He did not bite his tongue. His legs were moved up and down bending at the knee and then straightening. This fit passed off in a few minutes, and he immediately started to speak just as usual. He had two other similar fits during the afternoon and when I saw him in the evening he had quite recovered. He could speak quite well. Next day about 1. p.m. when I saw him he was going on well. There was no aphasia and I procured a specimen of his writing spontaneously and to dictation and to copy, from which it will be seen that he

- I. Wan Eggers
- II. 1, 2, 3, 4, 5
- III. I can write so
- day. HE
- IV. I am better
- V. I am better

- I. Voluntary ;
 II. Dictation of Figures ;
 III. Dictation of sentence ;
 V. Patients copy of IV.

Fig. XXII.

had quite recovered from his agraphia as well as his aphasia. See Fig. XXII.

About 2 p.m. he suddenly was seized with aphasia just as before. His jaws were clenched, he could not speak, he had no paralysis of any kind. This condition lasted till 3 a.m. (13 hours) when he again quite recovered, and he has remained all right till this date 15th April, 1896.*

Answering our 12 questions it will be seen that,-

1. He could hear sounds.
2. He could hear words.
3. He could understand words.
4. He could see objects etc.
5. He could see words.
6. He could understand written words.
7. He could not speak voluntarily.
8. He could not repeat words.
9. He could not read aloud.
10. He could not write voluntarily.
11. He could not write to dictation.
12. He could not copy.

It was therefore a very complete case of cortical motor aphasia. The most interesting fact about the case is its temporary character, and complete and sudden recovery. The first attack lasted at least six hours, the second thirteen hours. He had

* This patient was seized with symptoms of uraemia on the morning of April 26th. He had 1st a sort of fit and then uraemic asthma and died comatose on the morning of 27th April 1896. (W.B.)

albuminurea, and there was a distinct history of his having partaken freely the night before of nitrogenous food, but I must say that he had not the appearance of a case of uraemia. It is well known that there are some cases of aphasia produced by some forms of toxæmia and uraemic aphasias have been described by various authors. In the Lancet for April. 11th, 1896, a case is quoted which Dr Rendu physician of the Hôpital Necker, Paris; reported to the Association Medicale des Hôpitaux on March 27th, 1896, in which the patient had been seized with an apoplectiform attack. After regaining consciousness three symptoms remained, viz:- aphasia right brachial monoplegia and a systolic bruit at the base of the heart. Embolus was diagnosed, but a few days later the patient was seized with intense dyspnoea going quickly into Cheyne-Stokes respiration, the urine was scanty and albuminous, Phlebotomy was practised, the blood revealing the presence of 75. centigrammes of urea per litre, evidently it was uraemia. The blood letting improved his condition, the dyspnoea disappeared and the somnolence gradually diminished, whilst the vocabulary became more extensive. A month later no trace of either aphasia or monoplegia remained.

The case of diabetis insipidus, recorded by

Chouppe and quoted in the earlier part of this paper, where a dehydration of the blood produced aphasia, may also be referred to in this connection. Whilst therefore case VIII. in all probability was a case of uraemic aphasia very complete and sudden in onset and rapid and sudden in recovery analogous to some cases of uraemic amblyopia, which are sudden and complete in character and temporary in duration, still, a theory for which some evidence can be produced, is that the case was functional or hysterical in its causation. The patient had not the appearance of a uraemic patient, the fits were not uraemic but more like hysterical fits in character.

Hysterical motor aphasia due to loss of function in a cortical area have never however been described. All hysterical mute cases conform to the subcortical or infra pictorial motor type. We know that this case was cortical in type from the presence of agraphia. Taking all the evidence therefore in all probability, it was a case of aphasia due to uraemia.*

The following short note of a very temporary aphasia case may also be inserted here.

* The subsequent history of the case proved this supposition to be correct as the patient died from uraemia on April 27th/96 (nr)

Case IX. (Personal Observation.)

M. F. aged about 78, on three different occasions had been seized with sudden and temporary aphasia. On at least one occasion it had come on in the middle of a sentence. She stopped speaking, looked dazed and remained unable to speak for three or four minutes.

On one or two other occasions she found herself unable to speak. She had a peculiar dazed feeling, could see and hear sounds and words quite well, but could not utter any word. She has been troubled with weak action of the heart, otherwise she is in good health. I have not seen her in one of these attacks but have been summoned to her immediately after one. Recently she had one attack one evening and another attack on the following evening. The most feasible explanation of a case like this is, I believe, that there is a temporary disturbance of the circulation or interference with the nourishment of the cortex in Broca's convolution, so interfering with the function of the cortical area producing temporary suspension of function. Of course this case also might be classified under hysterical cases but in all probability there was some physical interference with the function of the psycho motor centre.

These three cases of Pictorial motor aphasia give a good clinical picture of the symptoms of this variety of motor aphasia, the 2nd one being the only one whose case was thoroughly investigated in all its aspects so as to ascertain the answer to each of the 12 questions although I have indicated sufficiently the symptoms in the other two cases to classify them in this group.

Passing now from cases of the Pictorial or Cortical motor groups to cases of III.

Infra pictorial or subcortical motor group.

We will see that this form is distinguished from the Pictorial form as I have previously indicated in this paper by the fact that the patient with this type knows how a word ought to be articulated, that is, he knows how many syllables it contains. He is also able to write voluntarily to dictation and to copy. Cases of this variety are not common as organic lesions but there is a functional form of speech disturbance, viz., Hysterical mutism which conforms entirely to this variety. Charcot (52) has described cases of this kind and Bastian (56) in his able work on Hysterical and Functional Paralysis endeavours to locate all Functional and hysterical Paralysis to particular parts of the nervous

system. Amongst others he locates Hysterical mutism or the Functional form of complete aphemia. He says (page 50) "it must be due to functional degradation occurring in the course of the internuncial fibres that pass between the glosso kinaesthetic centre in the posterior part of the 3rd frontal convolution and the motor centres for articulation situated in the bulb." In this class of cases patients are absolutely dumb that is they are voiceless as well as speechless, but they understand everything that is said to them, and can express their thoughts perfectly and with unimpaired facility by means of writing. Their intellectual faculties are moreover quite unimpaired." Again later, (p.53) he says, "I may repeat then my belief that this form of speech defect may be produced by damage to efferent internuncial fibres in any part of their course from the left glosso kinaesthetic centre to the articulatory centres in the bulb." These views of Bastian although propounded by such an able observer as well as clear writer I cannot altogether agree to - because as I think I have proved in an earlier part of this paper, the glosso-kinaesthetic centre is not in the posterior part of the 3rd frontal but in an area further back, viz., in the lower part of the ascending frontal and ascending parietal

convolutions. Case I. as also the case I quoted from Mills (28) proved distinctly that this was so and that the psycho motor speech centre was in the posterior part of the 3rd left frontal, whilst besides the centres for the movements of the tongue (the glosso kinaesthetic centres of Bastian) there were also in the lower part of ascending frontal and ascending parietal convolutions centres for adduction and abduction of the vocal cords, and centres for the movements of the lips, cheeks and lower part of the face. Mills case and mine proved that lesion of these areas, the executory motor centres or centres for the vocal and oral articulative mechanism produced paresis not paralysis of the muscles of the vocal and oral articulation on the opposite side. This is a dysarthria and not an aphasia at all. Lesion of the fibres from these centres to the bulb would produce the same symptoms. If the lesion was on both sides then of course there would be aphasia, but there would also be pseudo bulbar paralysis, that is paralysis of the oral articulative muscles. But in hysterical mutism there is no such paralysis. The patient can move his tongue, lips, cheeks, jaws, etc., and I believe also the vocal cords as usual, although he cannot do so to produce words. What is wanting is the proper

cell grouping in the vocal and oral articulative centres and as I have shown this arranging, co-ordinating or cell grouping in these centres, so that the correct movements in the muscles may be produced is brought about by the psychomotor speech centre in the foot of the 3rd left frontal. The position of a lesion either functional or organic which produces a complete infra pictorial motor aphasia must be therefore so that it cuts off the psycho motor centre from the vocal and oral articulative centres on the same and the opposite sides. Instead therefore of the interference being in the efferent internuncial fibres in any part of their course from the left glosso kinaesthetic centre to the articulatory centres in the bulb, as Bastian says, the interference must be in the fibres passing from the psychomotor speech centre (B Fig. XII) in the posterior part of the third frontal to the vocal and oral articulative centres of the same (C_3) and the opposite sides (C_1).

(196)

The following are two cases of Hysterical Mutism, which is as I have said the functional form of Infra pictorial motor aphasia.

Case X. (Personal Observation.) Complete motor aphasia, sudden in onset, gradual in recovery able to read, to write, and to understand spoken speech perfectly, in a highly intelligent working man, whose intellectual faculties were not in the least affected by the attack.

J.S. aged 37, married, admitted to Leith Hospital, November 5th, 1895. History, Five years ago whilst in a passion, patient fell down and lay insensible for about 20 minutes. When he wakened up he could only speak imperfectly. He could just use words but could not join his sentences. Within an hour the speech returned completely. His wife thinks that during that attack he could hear and understand quite well.

About a year ago he was kicked on the thorax and head by a horse, and lay unconscious for some time according to his wife's story, but according to his own he was not insensible at all. Since then till the present attack he has been healthy.

On November 5th, 1895, patient went out to work apparently in his usual health. In the forenoon he

had had some sort of vague feeling of illness, he felt out of sorts, had no pain, but felt cold and shaky. He told the manager that he would have to go home as he did not feel well. He was "all out of sorts" and unable to take charge of horses. He loaded his lorry at one street and delivered it at another. The last thing he remembers was asking a man to give him a lift with some casing. He was seen to drop down suddenly beside his horse. He lay unconscious for ten minutes, apparently no convulsions. Dr Langwill, Leith, saw him, and sent him to Leith Hospital. When Dr Langwill saw him he had regained consciousness, and had no motor paralysis, but was unable to speak.

State on admission.

Patient was brought in on a stretcher, but had quite the use of his limbs. He was a man of fairly good muscularity, and had no obvious morbid appearance.

Nervous system..

There was no paralysis, sensory or motor, of the extremities. Knee jerks normal, no ankle clonus. Pupils were equal and re-acted to light. Organic reflexes were normal. His intellectual functions appeared normal, but when he attempted to speak he seemed to get excited.

Speech, Motor. He was quite unable to speak, to answer questions, or to repeat words. On attempting to do so it was seen that his face gradually became flushed, the right side of his face moved, then first his right hand, and then his left hand, were moved gently, and then rather forcibly, so that sometimes he could be described as "striking out." His legs moved restlessly in bed, he raised himself a little off the pillow, and then fell back exhausted with a deeply flushed face, and taking deep breaths. This process was gone through whenever he made an attempt to speak, and it continued although in a gradually lessening degree, as he gained more power in speaking. It seemed to be an overflow of energy from the motor speech centre to the other motor centres, and brought to one's mind the efforts that a very bad case of stammering makes in his efforts to get out the proper pronunciation.

He was able to write without the slightest difficulty. In writing he moved his hand in a rapid excited manner. Asked his name, he wrote it quite well, as well as his address. He then wrote of his own accord, "I know well enough what you say, I am sensible." Later he wrote, "I cannot articulate." Shown a watch and asked what it was, he wrote, "Watch." Questioned as to time he wrote correctly, 4,25.

Sensory speech. There was no apparent impairment. He seemed to understand without any difficulty all that was said. He copied writing and

print quite correctly, He wrote to dictation quite correctly, wrote figures to dictation, and could count them up correctly.

156
459
346

961

he wrote down the answer correctly. He could tell the number of syllables in a word. Ophthalmoscopic examination revealed no abnormality. There was no albuminurea, nor sugar in the urine; there were a few uric acid crystals in urine.

6th November. Asked, "How are you to-day?" He answered with great effort "Better." Then he wrote, "I am all right if I could only get my speech." He answered, "Yes" and "No" with great difficulty to some questions. He wrote, "I can hear everything that goes on." On being questioned about his musical faculty he wrote, "I am no musician." He could not tell his own name in speech, nor repeat it after one, but he could repeat "Yes", and "No," and "Better," but with great difficulty. When he did get a word out, it came with a great explosion, and in a loud voice.

7th November. The following was the result of his attempts at naming the letters of the alphabet.

A + means he was able, an o he was unable to pronounce the particular letter below which it is.

a b c d e f g h i j k l m n o p q r s t u v w

o o + o + o o o + o o o + + + + + + + + + +

x y z

+ + +

g was pronounced with very great difficulty, and with a violent explosion, w also with great difficulty.

It will be seen that the first letters of the alphabet he did not produce so easily as the later ones; the further he went on, the better he spoke.

To-day patient said "Quin," the name of the nurse in the ward, and learnt since his illness.

When asked to say 1. 2. 3. 4. 5. He said one and two.

8th November. He said "Good morning."

"Do you like that book?" (A) "No," and wrote, "It is childish."

"Say childish." (A) "Child." Say childish.

(A) "ish"

"Say it again." (A) "Child - ish."

"How many syllables are there in "childish"?"

(A) "Two"

When asked how he knew the number of syllables, he wrote "I know how it should be articulated." Asked what he felt in trying to say for instance

"Quin", he wrote, "Exhaustion and a slight pain," pointing to left side of head just over Broca's Convolution. "How do you think the words get out?" He wrote "Some of them are coming away naturally."

(Q) "And the difficult ones? (A. in writing) "I have to force the articulation, the articulation is there."

The following was the result of his attempts to name the letters of the alphabet.

a b c d e f g h i j k l m n o p q r s t u v w x y z
+ + + o + + o o + + + + + + + + + + + + + + + +
g. w. and z. with great effort.

9th. Asked, "How are you feeling to-day?"

(A) "Well." "How did you sleep?" (A) "Rested well. I dreamt - that - I - was - preaching - t-to - the - whole - ward."

"What were you preaching about?" (A) "The l-o-o-ost lamb."

"Can you tell me your name?" (A) "No."

"Can you say it after me?" He did so after great effort.

Asked to name the alphabet the following was the result.

a b c d e f g h i j k l m n o p q r s t u v w x y z
+
g. w. and z. with great effort. Asked to read out of a book he read correctly. "The - door - flew -

open - as - doors - al - ways - do - when - there's
- a - boy - at - the - other - s-s-side.

There was a good long pause between the words. By the time he had reached do he put down the book exhausted, in a minute or two he continued "And - To (= Tom) s-s-s-sidy - rushed - in - almost - breathless." (last word with great effort) "I - am - strug - ger," "What? Struggling?" (A) "No." "Stronger?" (A) "Yes." He then wrote, "the articulation and words do not seem so far away to-day."

On 10th November he was speaking still better. Could use the whole alphabet, and could speak sentences, but words separated by a pause. On the 12th it was noted that he had now only difficulty with difficult test sentences such as, "British Constitution," and "My mother munches mushrooms."

On the 13th November he was dismissed with speech quite returned exactly ten days after admission. Dr John Wyllie Edinburgh kindly saw the case with me on the 10th November.

The notes of the case were taken by Dr Hill Buchan, House Physician, to whom I am indebted for them.

The notes of the case are so explicit that it is hardly necessary to say much about them. The patient was a very intelligent man, and as he was able to write, we were able to ascertain his own

opinion as to the cause of his mutism, and his feelings in attempting to speak. On several occasions he wrote "I cannot articulate." "The articulation cannot get out," - "I know how I should articulate a word," - "but I cannot get it out."

When recovering he wrote "that the words he could speak came away naturally", "but the difficult ones he had to force the articulation", "the articulation is there." He also wrote that speaking a word gave him a feeling of exhaustion, and a pain over the left side of the head (pointing to Broca's convolution).

These opinions and feelings of the patient are so much in accord with the opinions I have expressed with regard to the position of the functional derangement in hysterical mutism, viz., in the same region as an organic lesion which produces an infra pictorial motor aphasia, that I have recorded the notes of the case in full.

The extent of nerve fibres whose function is deranged is short, especially on the left side, where they pass only from the posterior part of the third left frontal, to the lower part of the ascending frontal and ascending parietal convolutions, the fibres passing from the third left frontal to the vocal and oral articulative mechanism of the right side have however, a greater length. As there was

no paralysis of the muscles of the tongue, lips, etc., there could not have been functional derangement of the fibres passing from the vocal and oral articulative centres in the lower parts of the ascending frontal, and ascending parietal convolutions on both sides to the centres in the bulb, which fact, as I have said, is contrary to the theory and opinion of Bastian.

The answer to the twelve questions in this case were,

1. He could hear sounds etc.
2. " " " words spoken
3. " " understand words spoken.
4. " " see objects, etc.
5. " " see words written and printed.
6. " " understand words written
and printed.
7. " " not speak voluntarily.
8. " " not repeat words.
9. " " not read aloud.
10. " " write voluntarily.
11. " " write to dictation.
12. " " copy.

Although he could not speak voluntarily, repeat words, nor read aloud, he knew how the words should

be articulated, that is the number of syllables, etc., the word contained. This, as will be seen is exactly what we have found to be the case in Infra pictorial motor aphasia.

The next case is one of the same kind, viz., a case of Hysterical Mutism. It was seen by my brother, Dr George Elder, whilst assisting me, and to him I am indebted for the notes of it.

Case XI. Hysterical Mutism for 18 hours.

C.D. a girl aged 15, employed in a dress maker's shop, was seen in Leith in February 1895. She was said to have fallen down in a fit about 6 hours previously, and had never been able to speak since.

Family History. Her father had tubercular joint disease, but none of the other children, seven in number had any disease. There was no epilepsy or other nervous affection in the family. All her mother's family menstruated very early, at about 12 years.

Previous Health. Very good. Began to menstruate before 13. Had recently very long hours.

Present illness. Came on quite suddenly when standing by the fireside speaking to others. She fell down on the floor, and her arms and body were thrown about freely for a few minutes, and then all movements ceased, but patient could not speak. It was impossible to make out from the history whether

the patient was conscious during the fit.

State on Examination. She was seen about 12 p.m. She was lying quite still in bed and quite conscious - she followed every movement with her eyes, and understood evidently everything that was being said, but when she tried to speak although the lips moved, absolutely no sound was emitted. Temperature was normal - pulse steady and normal, free perspiration. No sign of any weakness or paralysis anywhere. Heart sounds were quite good, and absolutely nothing abnormal could be made out on examination. The mother was told not to alarm herself, and not to make much to do about the patient, or to shew her too much attention.

She was seen again next day about noon. She had slept well and taken her food, and was quite sharp in every way, but had never uttered a sound. It was then explained to her that the voice was produced by two bands in the throat, and that these had got affected so that they could not be tightened, but that if they were tightened she would be able to speak as before. The fingers were then firmly pressed over the thyroid cartilage, the wings being pressed inwards, and she was told that she would then be able to speak. On being asked if she could she said, "Yes," in a clear tone, and she was able to answer quite clearly all the questions asked, and to

speaking quite freely. So far as known there has been no return of the mutism.

This case besides being a very typical one of Hysterical Mutism, is interesting from the method of treatment which was adopted in its cure. It is a method that I have very frequently adopted for the cure of Hysterical and functional aphonia, whether that aphonia was due to a previous laryngitis or came on in a hysterical subject.

I don't know who originally suggested this method, but it is one which I know to be practised by several physicians.

The patient is told that pressing the wings of the thyroid cartilage will enable her to produce voice and she is asked to say ā or ē, or ō, whilst the fingers press the cartilage. The result frequently is that voice is at the moment produced, and after the fingers are withdrawn she still continues to use her voice.

Hysterical mutism differs however, from Hysterical aphonia, in that in the former there is no voice or sound whatever produced by the larynx, or muscles of oral articulation, whereas, in Aphonia the patient can speak in a whisper, or a very low broken voice.

AGRAPHIA. GRAPHIC APHASIA.

In our study of the other forms of aphasia we have seen how frequently agraphia is an accompaniment of them.

1. We have seen that in Auditory aphasias, the patient is able to write spontaneously, although if the lesion is cortical, there is usually paragrammia in spontaneous writing, as there is also paraphasia in the patient's speaking. Auditory Aphasia cases, however, have agraphia to dictation, except the supra pictorial form, where the patient may be able to write letters and short words, but not long words or sentences to dictation because they have to be written mechanically without reaching the ideational centres.

The patient is usually able to copy writing when he suffers from auditory aphasia.

2. In visual aphasia if the lesion is infra pictorial, the patient is able to write voluntarily, and to dictation, but there are great disturbance in his powers of copying. He simply copies by tracing the letters as he would a drawing.

In Cortical Visual Aphasia on the other hand

the patient is not able to write spontaneously, nor to dictation, and he can only copy very imperfectly, as from a design - as the visual images of the words and letters are blotted out.

3 In Conduction Aphasias (Leitungsaphasie) the patient can usually write, that is form the letters, words, etc., by writing voluntarily and to dictation, but he shows paraphagia, that is, he writes the wrong words and letters, just as he speaks incorrect words "paraphasia." He is, however, able to copy.

Theoretically there are two forms of paraphagia 1st, the form due to paraphasia, where the incorrect word memory is raised in the psycho-motor speech centre. The patient in that case writes correctly the paraphasic word, but in the other theoretical form it is possible for the patient to write incorrectly the letters of a word which would be correctly spoken, or he might write a different word from the word he spoke, or he might trace incorrect lines in writing the letters. These would all be different forms of paraphagia.

4. In motor aphasia. If the lesion is infracortical there is no disturbance of writing.

If cortical the patient has agraphia to

voluntary writing, and to dictation, but he can copy at least as a drawing.

Is there any evidence to show that there have been any other forms of agraphia than those due to lesions of the other speech centres? Very little evidence, and certainly none convincing can be found in medical literature.

Exner (57) in 1881 produced some evidence to show that there was a special graphic centre in the posterior part of the second left frontal convolution, and this had been pretty generally accepted till in recent years. Déjerine (13) has done much to show that all the cases that have been recorded in favour of a special graphic centre could be explained in some other way than admitting the existence of that centre. He showed for instance that the well known case of Henschen (Margarita Anderson) besides having agraphia, had also word blindness and hemianopsia, and at the post mortem besides having a lesion in the posterior part of the 2nd left frontal convolution, there was another lesion in the angular gyrus, the latter lesion being quite sufficient to account for the agraphia independently of the other.

The case of Bar (58) can be explained in a

similar way, there had been a motor aphasia along with the agraphia. These are the only two post mortems, which I can find that might be produced as evidence of a special graphic centre. There are, however, a good many cases where patients have only shown the symptoms of agraphia clinically, but most of these cases had also showed symptoms of the other forms of aphasia, at some time or other. Such are the cases of Charcot and Pitres (19) quoted by Dèjerine (17) and Wyllie (20). In Charcot's case there had been motor aphasia some time previously, and in Pitre's case, there was hemiplegia and right homonymous hemianopsia, but no word blindness nor motor aphasia. Although he had almost complete agraphia with the right hand, he had trained himself to write with the left.

There was no post mortem in either case, but the history shows that other parts than the graphic centre must have been at one time involved so that they are not at all conclusive as to the separate existence of a graphic centre. We must take it therefore that no conclusive evidence can be got as to the existence of a graphic centre from a study of cases of agraphia that have been recorded. That being so what evidence can be got from a theoretical study of the

subject of writing? In learning to write the individual simply traces the lines as he sees them before him in the copy, but through practice he becomes more expert in the particular movements necessary to form the letters and their combinations into words. When writing is done from memory voluntarily and to dictation the visual image of the letters is raised in the visual memory, and to a great extent the writing is traced from the copy perceived in the visual memory, just as it would be if the copy were placed before his eyes. On this theory, then, the movements of the hand in writing are directed or guided by the word seeing centre, in other words the cell grouping in the centres for the hand and fingers is directed, or co-ordinated from the word seeing centre in the left angular gyrus. But whilst this may be so for the ordinary outlines of letters, there is, however, something more than this in the individual who has been trained to write.

Where are stored the memories for the fine movements and delicate touches which produce the peculiar shapes and flourishes in the letters of the expert writer?

Why are letters formed so differently by different individuals? Why are little peculiarities in an individual's writing reproduced over and over again,

so that there is no mistaking one person's writing from that of another, although we have all learned to write from the same, or very similar copy, and raise always much the same visual letter images in our word seeing centre? We not only are not conscious of raising in the visual memory, the particular images of the letters as written by ourselves, but many of us if asked to say how we usually wrote a particular letter, would not be able to tell until we had practically done so by writing it. Where then are the movements which produce these delicate lines etc., guided from? Are they guided from the visual word centre, or from a special centre in the immediate neighbourhood of the centres for the movements of the hand? I believe that at first writing is done entirely from copy, but that the memories of the muscular movements for writing are stored in the neighbourhood of the motor centres for the hand. Writing, it must be remembered, is only one of many of the different accomplishments for which the hand can become specialized, and the same muscles are used for all of them although in different combinations. Take for instance the very delicate movements of the musician playing such a musical instrument as a piano, a

mandolin, or a violin, or let us take the movements necessary for expert typewriting, the movements necessary for painting, the movements necessary for the handling of a knife by an expert surgeon, the movements necessary for the handling of a club by a golfer, or the cue by a billiard player, or the movements necessary for the doing of any work by a skilled workman. Are not impressions of these skilled movements conveyed by the tactile nerves, and nerves of the muscular sense, to the nerve cells in the cortex to be there stored in the memory to be reproduced again when necessary? Is it not reasonable to suppose that certain cells get specialized for the storing up of the memories of these special movements, so that they can be reproduced at will, and is it not reasonable to suppose that these cells act by bringing about the necessary cell grouping in the motor centres for the particular part of the body, where the particular muscular movements are produced? On this theory then, there are certain specialized centres in the neighbourhood of the motor centres for the hand, and one of these specialized centres is that for writing.

If for a moment one considers all the movements necessary for writing, and if it be granted that a

nerve centre varies in size in direct ratio to the number of combinations necessary to produce the movements for which it has become specialized, then the graphic centre need not necessarily be a large one in order to produce all the letters and figures and all their combinations. It is not necessary to have stored in a graphic centre the memories of the graphic images of words, because we do not write words as words. We write letters and combine them so that words are produced. All that is necessary, therefore, is to have the graphic images of all the letters in their capital and small form, and all the figures and other signs used in writing, and the graphic images of the appendages to the letters and figures, such as the lines connecting them, special flourishes etc. The graphic centre is enabled to combine these letters into words by means of the psycho-motor speech centre, and the auditory word centre because as I have already stated, in an earlier part of this paper, when we write there is a process of silent articulation always going on, the articulatory image as well as probably the sound image of the letters and words raised in the cerebrum. Hence the necessity of having both the psycho-motor, and the auditory sound centre unaffected

if correct writing is to be produced. This is the reason that a cortical motor aphasia always produces agraphia.

If one considers this it will at once be seen that there are not very many graphic image memories required altogether. We are all in the habit of writing each letter in a particular form and if we wish to change that form we at once call in the help of the visual centre in a very decided manner, and trace the new form of the letter as we would from a copy. Take even our attempts to print with a pen or pencil all the letters of the alphabet, or our attempts to write the letters of a language whose letters are unfamiliar to us. We simply trace them as we would a map or design. If we have not the form of the letters in a copy before our eyes, we raise them in the visual memory and we mentally see them. And not only can we write letters of any language or any shape by simply tracing them in this way, with the right hand, but we can do the same thing by means of the left hand, although not quite so well as with the right.

The only difference is that our right hand is used for almost every action, and the movements of the right hand are more under the control of the will, and

better co-ordinated than those of the left, from more constant use, and from the fact that it is trained to hold a pen and to draw lines, curves, etc., although not the exact form of the strange letter it is attempting to write. Although such letter as a whole is strange, parts of it are familiar, so that the right hand, which is accustomed to draw lines etc., is more expert at the new letter than the left, but the expertness with which the letter is written is a long way behind the expertness with which a familiar letter is written.

Not only can letters be traced as from a copy by the left hand, but the same thing can be done by a pen between the teeth, by one attached to the leg, or the elbow. This, I believe, is brought about by the guidance or direction of the visual centres where are stored the memories of the forms, shapes etc., of objects.

But how different is this from writing! Is there much resemblance between the writing as written by the right hand, and that written by the left?

If one knew a man's handwriting, as for instance, his signature, would it be possible to tell his signature if he wrote with his left instead of his

right hand, and yet he has got the same muscles in the left hand as in the right, and has got the same visual centres to guide it. There would probably be slight resemblances, but this would result merely from having the form and shape of the letters of his signature in his visual memory when he was tracing it.

The fact is we are forced to the conclusion that there must be stored in, or in the neighbourhood of, the cortical centres, for the movements of the right hand, special cells whose function it is to do the cell grouping or co-ordinating of the movements necessary for writing in the true sense of the word. These cells form, therefore, the psycho-motor graphic or the special graphic centre, but the area where they are, need not necessarily be anatomically separable from the centre for the hand, so that it is quite possible that no pathological lesion could blot out those cells and at the same time, leave the ordinary motor centres for the hand intact. The fact that no really well authenticated case has been produced, where it could be said with certainty that the psycho-motor graphic cells were alone involved, shows that in all probability these cells are not pathologically separable from the centres for the movements of the

hand in the middle of the ascending frontal and ascending parietal convolutions. The position the graphic centre has usually been considered to be located in, is a little further forward viz., in the adjoining part of the 2nd left frontal convolution.

It will be well, however, for Clinical observers to remember that if this graphic centre was alone involved without the movements of the hand being involved, and without the visual word centre being involved that there would not be loss of the power of writing.

Writing could still be performed just as we are able to write with the left hand, or with our foot, in other words the patient would be able to trace letters and words, but what would be lost would be the power to write in his usual handwriting, and in a rapid and expert manner.

This fact has not been sufficiently borne in mind by Clinical observers, so that we constantly see notes taken as to whether a patient was able to write or not, without paying particular attention as to whether he wrote in his usual handwriting or not.

A practical test for this would be to ask the patient first to print a sentence, and then to write a sentence. It would be found that probably he would

be able to print the letters quicker and better than he could write them. This results from the fact that letters as used in print are more distinct in their outlines, easier raised in the visual memory, and easier copied as one would a drawing, than letters as written. The movements necessary for their production are not so intricate and complicated, as those for writing, the lines being straighter and not having so many curves. I refer here to plain Roman printing type.

This fact I have noticed very distinctly in cases with *agraphia*, due to word blindness, as in Case VI, where there was a very considerable difference in the facility with which he copied plain printing from the difficulty with which he copied manuscript especially if the letters had any appendages, flourishes, or any little peculiarity of form. In attempting to copy the word North as written, he looked long at the loop on the N, and then made several attempts to write it. He seemed in great difficulty as to how it was to be added to the rest of the N.

This, I think, is brought out by a study of his writing, Fig XXI. Normally we all write manuscript

letters more easily, and better than we draw printed letters, whilst in graphic disturbances, whether from lesion of the visual word centre, or graphic centre, we draw printed more easily and better than manuscript letters.

In a former part of this paper I theoretically sketched three different forms of Graphic Aphasia, due to lesion of the graphic centre. As I have now stated no true authenticated cases of these lesions have been published, and therefore it is unnecessary to say a great deal here with regard to these forms. They are,

1. Supra pictorial graphic aphasia.
2. Pictorial graphic aphasia.
3. Infra pictorial graphic aphasia.

Although none of these forms have been described there is little doubt that a lesion which destroys all the centres for the movements of the hand, the specialized as well as the ordinary motor centres, that is a cortical lesion producing paralysis of the right hand, can be scientifically considered to be a case of graphic aphasia. Although all the movements of the hand as well as the movements necessary for writing are rendered impossible, still the patient looked at from the aphasic point of view suffers from agraphia.

If the lesion existed independently of the true graphic centre, he would have infra pictorial agraphia, if the true graphic centre was also destroyed he would have pictorial agraphia. He is probably able to write with the left hand, but this writing is simply by tracing or copying until he gradually educates the right hemisphere, and specializes another centre there. There are, therefore, many such cases of agraphia if these cases be accepted as agraphias, as I hold they are, but as I have said there are none in medical literature where the centres for the ordinary movements of the hands escaped, and the graphic centre alone, or the connecting fibres from it to the centres for the hand were involved in a lesion. Much also might be said in favour of the view that the case of Pitres (19) quoted by Déjerine (17) and by Wyllie (20. 358) was a case of Supra-pictorial graphic aphasia, that is a lesion in the fibres between the psychomotor speech centres, and the psycho-motor graphic centres, and not a lesion of the graphic centre alone, as has been frequently claimed.

A lesion in this position theoretically must occasionally occur, whether we hold there is a true graphic centre, or only a centre for the movements of

the hand, and the existence of this lesion probably accounts for those cases of motor aphasia with agraphia in which the latter symptom persists after the patient has recovered from the motor aphasia. These cases are not considered by Déjerine to be true agraphia cases, but to be simply the remains of the motor aphasia. I think, however, that theoretically as well as clinically they can be truly classed as Suprapictorial graphic Aphasias. (Lesion of B.D.)

MIRROR WRITING

Before passing from graphic aphasia, it is necessary to mention a peculiar symptom displayed by some patients who have to write with the left hand whether they have aphasia or not. They write from right to left instead of from left to right. as all Europeans do. The writing can be easily read by holding it up before a mirror. Some patients who display this symptom are not aware of their peculiarity. The exact significance of the symptom has not been made out, but it has been suggested that from right to left is

the natural way to write with the left hand. In some languages it is the proper way to write, as for instance Hebrew, but the Jews were not left, but right handed. Many cases showing this peculiarity have now been described, but its significance, as I have said, has yet to be ascertained.

VI. DISTURBANCES OF THE MUSIC FACULTY; AMUSIA ETC.

We have now considered all the different varieties of aphasia, and have been able to localise the different varieties to particular parts of the Cerebral Hemispheres. I wish now to consider shortly another faculty very closely allied both theoretically and clinically to the speech faculty, viz., the faculty of music.

Music can be received, retained or stored up, and produced by the cerebral hemispheres just as speech is. It is received by the auditory and the visual route, and is produced by the vocal and oral articulative mechanism, and the hand just as speech is.

But there is one great difference between speech and music, viz., that whilst in written speech the hand is used for writing, in music the hand is used for writing notes, but to a much greater extent for the production of music by playing instruments which have been devised for producing music. And it is not only one hand that is used for this production of music, but sometimes two, sometimes the mouth in addition, and sometimes the feet according to the particular instrument that is played.

From what I have previously said then it will be seen that it is necessary to suppose the existence of specialized centres in the neighbourhood of the ordinary centres for the movements of the particular part of the body used in playing. The music faculty must be therefore quite as complicated a mechanism as, if not more so than, the speech faculty.

But it is only a few who have educated their cerebral hemispheres to produce music in various ways. All have not trained voices, although all can produce a musical voice of some kind. All have not learned to write musical notation, any more than all have taught themselves to read it, and many have never learned to play a musical instrument. The part of the musical

mechanism which is probably most universal is the faculty of hearing it. Probably almost every one hears music as music, but there are enormous differences in the powers which people possess, not only in appreciating it, in the sense of its giving pleasure, but in the powers they possess of recognizing its pitch and other qualities. In other words people differ in "having an ear" or not "having an ear" for music.

The music faculty is very closely associated with the speech faculty, and disturbances of the one are very apt to be accompanied by disturbances of the other. Although closely associated they appear, however, to be quite distinct, and the centres appear to have quite a distinct localisation in the hemispheres, because we find some cases of aphasia without Amusia, and some amusias without aphasia.

In Case IV. the case I have described of Conduction Aphasia although the patient had a considerable amount of word deafness, his hearing for music was quite unaffected. He sat for hours listening to a piano, and the slightest mistake in a note, was at once detected.

In Case II, a case of word deafness, there was

a peculiar derangement of the music faculty, viz., what I have called "a hallucination of melody" where the patient heard distinctly a popular song ringing in her head, and was able to reproduce it by whistling.

The whole question of Amusia in its relation to aphasia has recently been very carefully investigated by Professor Dr. J. G. Edgren (62) of Stockholm, who has from medical Literature on aphasia collected 52 cases and shown the state of the musical faculty in each case. Many of these cases are most interesting ones, and taken altogether they show the most wonderful varieties of symptoms but Edgren is able to classify them into three groups.

I. Comprising cases 1 to 24 where there was aphasia in some form present but no amusia.

II. Comprising cases 25 to 46 where there were present one or more forms of amusia and one or more forms of aphasia.

III. Comprising cases 47 to 52 where there was present some form of amusia but no aphasia.

From these it will be seen that whilst in a large number of cases (little less than the half) both amusia and aphasia were present in the same case and

in a large number (nearly a half) some form of aphasia was present without any amusia that there were very few cases indeed (6 out of 52) where there was amusia without some form of aphasia.

These results and a careful study of each individual case, shews that although the centres for music are near the speech centres still they are not exactly in the same position. Case 52 was one of Edgren's own observation, and was a case of tone deafness which had word deafness also at first, but the word deafness disappeared leaving only the tone deafness.

At the post mortem a lesion was found in the anterior two thirds of the first and anterior half of the second temporo-sphenoidal convolution, parts that were also included in the lesion in a case of Bernard's with tone deafness, which he also quotes.

He therefore concludes that that is the position of the music hearing centre, viz.- immediately in front of the word hearing centre.

The following is a summary of the conclusions which he draws from a consideration of the whole question of amusia.

1. The musical faculty just as the speech

faculty can be entirely or partially destroyed from pathological processes of one kind or another, and when partially destroyed the faculty can be resolved into its different components whereby special forms of amusia arise.

2. The different forms of Amusia possess a certain degree of clinical independence both in their relationship to one another and in their relationship to aphasia.

3. The Clinical forms of amusia appear to be analogous to the Clinical forms of Aphasia, and are frequently but not necessarily accompanied by the analogous forms of aphasia.

4. Amusia can be present without aphasia, and aphasia without amusia.

5. It is probable that the special clinical forms of amusia - at least certain of them - possess a certain anatomical independence, that they can be localised in the neighbourhood of the places where the corresponding forms of aphasia are considered to be localised, but nevertheless not in the identical locality.

6. For a special form of Amusia viz., tone deafness, it is highly probable that the localisation

appears to be in the first or in the first and second temporal convolution on the left side in front of the area, injury to which produces word deafness.

These are the conclusions of Edgren from a study of nearly all, if not all the cases in medical literature from which it will be seen that in all probability the musical faculty as well as the speech faculty is situated in the left cerebral hemisphere only, and although the auditory centres for ordinary sounds are in both hemispheres, that the specialized centre for the recognition of musical tones, etc, as well as that for words, is in the left hemisphere only.

Disturbances of the Faculty for Interpretation
and Production of signs. (Pantomime Language.)

Amimia, Paramimia, etc.

It has been found that some patients although aphasic understand and produce pantomime or gesture language quite well and on the other hand some have lost all knowledge of gesture language (Amimia.)

Some again put a wrong interpretation on gestures or may not produce the correct gesture as for instance they may nod their head instead of shaking it when they wish to say No, and vice versa. This is called paramimia. Some of the cases I have recorded possessed gesture language perfectly although very markedly aphasic as cases II. and III. the latter of which cases conversed for seventeen years almost entirely by signs. Probably the centres for interpretation of gesture language are in the occipital lobes in both hemispheres and not only in the left as the written word centre is, and, therefore are not so often entirely destroyed.

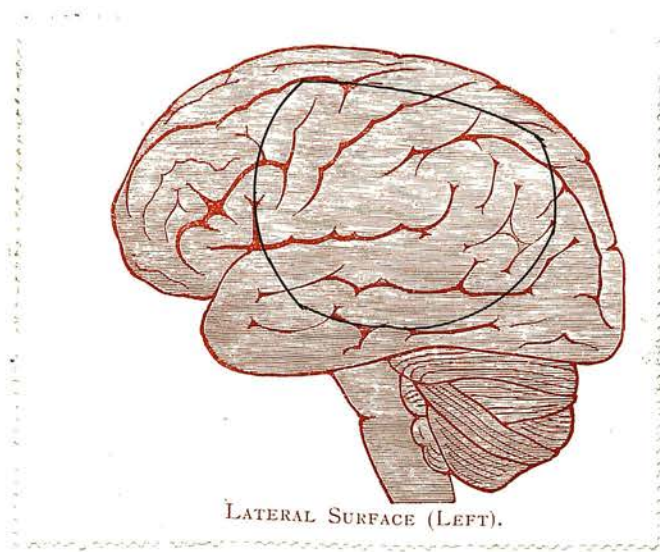
VII. Aphasia from a Surgical Point of View.

I have in several parts of this Thesis pointed out how important it is for the Surgeon to possess a very exact knowledge of aphasia. As I said in my introductory remarks, the subject has not occupied so important a place in our text-books of surgery as it ought to occupy. In recent years cerebral surgery has come very much to the front, owing to the most encouraging results got by such brilliant operative Surgeons as MacEwan of Glasgow and Victor Horsley of London. Now the first essential for a surgeon who has to do with cerebral disease in whatever form is to locate exactly the lesion that is causing the symptoms which he wishes to relieve.

Of all the symptoms which cerebral lesions cause there are none more important and few are so important as those which produce disturbances of the speech faculty.

If we look for instance at the superficial area alone of the left hemisphere it will be seen that if we draw a boundary line which would include all the speech centres within it, the area so included would occupy more than a third and very nearly a half of the whole superficial area, of the outer surface of the left hemisphere. See Fig.XXIII.

A lesion of any size within that area would be



The dark line encloses the Area
within which lesions of any size
are apt to produce disturbances of Speech

Fig. XXIII.

very apt to produce some disturbance of speech, the disturbance varying as we have seen according to the precise position of the lesion. And not only do cortical lesions produce speech disturbances but as we have seen subcortical lesions and lesions in the white matter of the cerebrum also produce speech disturbances and these disturbances vary according to the precise position of the subcortical lesion. I have not drawn that boundary line so as to include the whole of the occipital lobe which might fairly be included as within the speech area because as we have seen, lesions in the occipital lobe almost always produce some form of word blindness or symptoms associated with word blindness and the subject of aphasia. The importance of the consideration of aphasia therefore to the surgeon cannot be over estimated.

Many affections which truly belong to Surgery and not to medicine are apt to occur in the cerebral hemispheres. I have no intention of entering into a description of all Surgical cerebral affections but I shall only mention briefly a few of the surgical affections which may produce aphasia symptoms. Perhaps the most important of these are those due to traumatic causes. Accidents to the head of various kinds. Blows producing concussion, fractures, haemorrhages, inflammation and

abscesses. It is very essential that these be accurately localised.

And it need hardly be also emphasised how important it is for cerebral tumours to be localised. The subject of the removability of these has been very much discussed in recent years and although probably the proportion of those entirely removable with a good permanent result is not so great as could be hoped for still many have been removed most successfully. Another class of cases where considerable success has been obtained are cases of epilepsy, either Jacksonian or more general epilepsy, due to a local cause such as chronic meningitis, with adherence of the membranes to the cortex, or exostoses or spicules of bone due to old traumatic causes which give rise to irritative symptoms.

The importance of the subject being recognised can any method be laid down for the surgeon for the investigation of a case producing aphasic symptoms?

I take it that the three most important questions that a surgeon has to answer when he investigates a cerebral case are these.

1. What is the nature of the Lesion?
2. Where is it located?
3. Is it Cortical or Subcortical?

Having answered these questions he is in a position to decide as to how the case is to be treated, whether by operation or otherwise. Now although a study of aphasia may not give him very much help to decide as to the first question an accurate knowledge of the symptoms produced by each variety of aphasia is most essential in order that the second and third questions may be accurately answered. I do not for a moment wish to state that a study of aphasia would give no help to decide the first question vizt., the nature of the lesion. Indeed in one of my cases, Case VI. when the diagnosis lay between, multiple cerebro-spinal sclerosis, general paralysis, and pachymeningitis, the fact that the patient had certainly a cortical word blindness went a very long way to show that the cause was a pachymeningitis. This of course was not a surgical case but the same argument applies to surgical cases, as it might have been a surgical case.

But whilst a knowledge of aphasia may not give much help in deciding as to the first question; in cases of left cortical affections it is very important in order to answer the second and third.

II. In affections of the right hemisphere a lesion might possibly be so situated that the absence of certain aphasic symptoms might give the

indication as to which hemisphere the lesion was situated in. These cases cannot nearly be so numerous as the cases, where the positive evidence of aphasia necessarily locates the lesion on the left side, except of course in left-handed persons.

Having therefore ascertained that there is present some speech affection the next thing that has to be ascertained is as to which of the five great groups of aphasia cases it belongs to, whether Auditory, Visual, Motor, graphic or conduction.

Now that can easily be ascertained by getting an answer to each of the twelve questions I have previously drawn out.

These questions however include almost the complete investigation of each case, and it is sometimes not very easy in surgical cases getting an answer to all of them.

By studying the "Table of Summary of Results" which I have previously drawn up, (page 94) it will be seen that there is a certain symptom common to all auditory cases vizt. (question 3) he cannot understand words spoken, a symptom common to all Visual cases, (question 6) he cannot understand words written, a symptom common to all Motor cases, (question 7) he cannot speak voluntarily and a symptom common to all graphic cases (question 10) he cannot write voluntarily.

If therefore it be ascertained that a patient cannot understand what is spoken to him, he suffers from some form of Auditory aphasia or word deafness.

If he cannot understand written or printed words he suffers from some form of visual aphasia, word blindness.

If he cannot speak voluntarily he suffers from some form of motor aphasia and if he cannot write voluntarily - he suffers from some form of graphic aphasia.

If he can understand spoken words, and written words and can speak voluntarily and write voluntarily but shows imperfections in these and especially if he shows paraphasia and paragraphia he suffers from Conduction Aphasia which includes Type IV. and Type XI.

It is very essential that the questions should be answered in the order given above because he may for instance not be able to understand written words, because he cannot recall the auditory image of the word, owing to his having auditory aphasia and he may not be able to write because he has visual aphasia or motor aphasia. It is therefore taken for granted that the varieties which precede have been ascertained to be excluded before a diagnosis is made of any particular variety.

If he has some form of auditory aphasia the

lesion must involve the posterior half of the first left temporo-sphenoidal convolution and if he suffers from visual aphasia the lesion must involve the posterior part of the left supra marginal convolution the angular gyrus, or occipital lobe. If motor aphasia, the lesion must be in the region of the posterior part of the third left frontal convolution, and if graphic aphasia alone, without any other form of aphasia, the lesion must be in the second left frontal, or the motor centres for the hand across the middle of the ascending frontal and ascending parietal convolutions.

If he suffers from Conduction Aphasia the lesion is probably in the neighbourhood of the Island of Reil and the Sylvian fissure.

III. Having therefore ascertained the locality of the lesion, the next point, and a very important one it is for the surgeon, is to ascertain whether the lesion causing the aphasia symptoms is in the cortex or is in the fibres under the cortex.

The answer to this question must very frequently decide the further question, vizt. Can the cause of the symptoms be removed by surgical interference?

Whether the lesion is cortical or subcortical ought therefore to be ascertained and I hold that it is not only possible but not very difficult

ascertaining this unless the case is a complicated one. Limited lesions producing symptoms are easiest located but although in many cases one would not be able to say exactly how much of the cortex or of the fibres leading to the cortex or away from the cortex were involved in each case, still one can say with a considerable degree of certainty that the cortical region of a particular area is involved, or is not involved, in the lesion.

Case VI. is a case in point where there can be little doubt that the angular gyrus was involved in the lesion probably a pachymeningitis, however much more of the cerebral cortex may have been also affected. Also in case III. where the diagnosis was verified by the post-mortem one could say with certainty that the cortex of the first temporo sphenoidal and of the angular gyrus, was affected however much more. The post-mortem revealed that not only these regions but nearly the whole of the temporo sphenoidal lobe was also destroyed. How therefore are we to ascertain a cortical from a subcortical lesion in each of the four varieties.

In Auditory Aphasias if the lesion is subcortical the Auditory word centre not being destroyed, the patient cannot hear words nor understand words spoken but he can understand written words and can read aloud, whereas in cortical auditory aphasias

with the word hearing centre destroyed the patient also cannot hear words nor understand words spoken, but in addition he cannot understand written words nor read aloud.

This is because as I have previously pointed out it is necessary to revive the auditory images of the words in order to understand written words (that is, read intelligently) and to read aloud.

The lesion producing a subcortical auditory aphasia must be a very limited one, as the course of the fibres from the ordinary auditory centre on the left side to the special auditory centre (the word hearing centre) must be a very short one and it is necessary to interrupt these fibres as well as the fibres from the opposite side in order to produce a subcortical auditory aphasia.

The cases producing subcortical auditory symptoms alone are therefore very few - and most auditory aphasia cases are also cortical and are therefore situated in the posterior half of the first temporo sphenoidal convolution. In connection with auditory aphasia it is necessary to remember also that amnesia of nouns may be a marked symptom and in that case the naming centre is involved which as I have shown is in all probability a little lower down than the auditory word centre vizt. in the third temporo sphenoidal convolution. If the

patient showed a marked difficulty in naming objects at sight probably the lesion would be subcortical or deep in the substance of the posterior part of the temporal lobe.

If the patient showed tone-deafness, the lesion probably would also involve the anterior two thirds of the first and anterior half of the second temporo-sphenoidal convolutions.

In Visual Aphasias: If the lesion is cortical, the patient in addition to being word-blind is not able to write voluntarily or to dictation. The part involved by such a lesion is the angular gyrus and posterior part of the supra marginal convolution.

If the lesion is subcortical, the patient is word blind but can write voluntarily and to dictation. He copies simply by tracing, he has usually also hemianopsia and hemiachromatopsia. The lesion producing this may be subcortically to the angular gyrus, but a lesion in the substance of the occipital lobe or in the cortex of the occipital lobe but passing in sufficiently far to interrupt the fibres from the visual centres both of the same and the opposite side might produce it, so that one could not be certain of the extent of a lesion producing an infra pictorial visual aphasia. All that can be said is that it is situated in the occipital lobe.

In Motor Aphasia. If cortical the patient would not be able to speak at all except a very few familiar words, such as yes or no, - Such a lesion would be in the posterior part of the third left frontal convolution, and if it extended further back vizt. into the lower parts of the ascending frontal and ascending parietal convolutions he would show the symptoms of dysarthria or paresis of the muscles of oral articulation of one side.

If Subcortical Motor Aphasia. The patient would not be able to speak but he would be able to indicate how a word should be articulated, that is the number of syllables it contained by pressing the hand once for each syllable. (L'expérience Proust-Lichtheim.) Such a lesion is very limited in area if the only symptom, because if it extended further back to the subcortical region below the lower parts of the ascending frontal and ascending parietal convolutions dysarthria would result just as in a cortical lesion of these same areas.

A cortical cannot be distinguished from a subcortical lesion of the lower part of the ascending frontal and ascending parietal convolutions, a point which Case I. and Mill's case distinctly proved. It shows that lesions on the left side, producing dysarthria without aphasia can be sometimes cortical, a point of very great importance to the surgeon and

one which has not previously to the description of these two cases been pointed out.

In Agraphia Cases pure we have not sufficient clinical evidence to show the difference between cortical and subcortical cases but theoretically a cortical case would not know how to write in his usual handwriting; whereas a subcortical case would know how it should be written, but would not be able to do it. It is also to be noted that both visual and motor cortical cases are agraphic and that auditory cases are agraphic to dictation. If the patient's right hand is paralysed or has agraphia pure the lesion is in the posterior part of second frontal or across the middle of the ascending frontal and parietal convolutions.

I think I have said enough to show how the different lesions causing aphasia can be pretty accurately located in the hemispheres, and if these lesions are removable, it is now, since so many different methods of cranial topography have been devised, a very easy matter to locate exactly on the outside of the cranium the position of the lesion so that operative procedure may be undertaken for the relief of the symptoms.

VIII. Summary of Thesis.

I have no intention of drawing any lengthy conclusions by recapitulating what has been already

stated, at I am afraid great length, in the body of the Thesis.

I have endeavoured to give an outline of the mechanism of speech as ascertained by a theoretical study of it, and by the experiments which disease has performed for us in the various forms of aphasic disturbances - I have shown that our knowledge of speech disturbances has gradually grown from the time when they were all classified together and believed by Broca to be due to lesion of one area until now when we have differentiated five main varieties of aphasia, vizt. Auditory, Visual, Motor, Graphic and Conduction.

Of these five varieties I have sketched three Types of each of the first four, vizt. Infra-pictorial, pictorial and supra-pictorial and two of the fifth, vizt. Inter-pictorial, Auditory-motor and Inter-pictorial visual-graphic. I have brought forward eleven different cases from my own practice, ten illustrating six of these different types and one a case of dysarthria.

Case I. was a case of Dysarthria.

" II. and III. were cases of Pictorial Auditory Aphasia.

" IV. was a case of Conduction Aphasia.

" V. was a case of Infra-pictorial Visual Aphasia.

Case VI. was a case of Pictorial Visual Aphasia.

" VII. VIII. and IX. were cases of Pictorial Motor Aphasia.

" X. and XI. were cases of Infra pictorial Motor Aphasia.

I have pointed out the distinguishing features of each of those cases and the conclusions, many of them new, to be drawn from them - I have shown how best to investigate an aphasia case and how the one type differs from the other. The position of the lesion causing each variety and each type has been indicated and illustrated by post-mortems of my own and others taken from medical literature. I have also devoted some attention to disturbances of the faculty of music and shown how closely related these disturbances are to disturbances of speech.

Finally I have shown how important to the surgeon aphasia is as a diagnostic symptom and how necessary it is that he should carefully differentiate the different types.

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